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Skin and Venereal Diseases
Nervous and Mental Diseases

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SKIN AND VENEREAL DISEASES.

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NERVOUS AND MENTAL DISEASES.

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SKIN AND VENEREAL DISEASES.

CHAPTER I.

CONSTITUTIONAL RELATIONS OF SKIN DISEASES.

The growing tendency to take into account the *non-specific results of syphilis and tuberculosis,* from the diathetic standpoint, is manifest in many recent discussions of the dermatoses.

W. A. Hackett and N. E. Aronstam classify[1] the complementary dermatoses of syphilis into: First, morbid conditions of the dermic glandular appendages which are divisible into (a) anomalies of function, (b) structure anomalies; second, inflammatory states of the skin; third, cutaneous neuroses; fourth, undefined ephemeral dermatoses.

The perspiratory glands are never affected to any great extent. The sebaceous glands bear the brunt of the process and exhibit either secretion derangement or actual pathologic states. Seborrhea sicca is more prevalent than seborrhea oleosa and may manifest itself years after when all syphilis has been eradicated. The duct of a gland is more frequently affected by pathologic change than the racemose extremity. Acne results; very intractable in treatment which recrudesces after temporary abatement and is attended by severe subjective symptoms. It is apt to occur during secondary defervescence, is not influenced by specific treatment, but yields to the internal administration of ichthyol and its local use with adrenalin solution. The hydradenitis destruans suppurativa of Politzer[2] attacks the sweat glands. It is characterized by the appearance of hard painless nodules in the substance of the corium which grad-

(1) Medical Age, Feb. 25, 1905.
(2) Jour. of Cut. and Genito-Ur. Dis., January, 1902.

ually increase and eventually become yellow tubercles. Capillary rupture and serum transudation gives these an hemorrhagic aspect. The transudate undergoes retrograde metamorphosis into pus. Molecular disintegration results. The lesion opens and finally forms a pigmented somewhat depressed scar. The disease is very intractable but at times disappears spontaneously. Specific medication is without effect. Eczema complementing syphilis is only more angry in appearance and attended by more marked exudation. Ischiorectal eczema madidans rubrum frequently occurs of a type previously described[1] in the Year Book. Dermatitis, usually manifested as a gangrenous type occurring upon a parasyphilitic integument, is a most intractable and dangerous dermatosis. Erysipelas is similarly dangerous and often has a fatal termination like gangrenous dermatitis.

Dermatoneuroses are the most predominant of complementary luetic dermatoses. They are an expression of disturbance of vasomotor equilibrium, either peripheral or central. Vaso-constriction is partially or totally abolished; the peripheral vessels dilate and allow serum transudation which raising the epidermal layers, creates the pomphi or wheals so characteristic of these dermatoses. In 75 per cent of the cases they result from absorptions of toxins from the alimentary canal arising from defective digestion or from albumin decomposition or else they result from vasomotor ataxia coincident with a general neurasthenic tendency. Urticaria is of very frequent occurrence, invading at times the entire surface of the skin and causing marked subjective symptoms. Urticaria pigmentosa, limited to the trunk and upper extremities belongs to this group. Herpes next to urticaria is the most frequent complementary dermatosis of syphilis. Herpes labialis is very intractable to treatment and has a marked tendency to recurrence. Herpes progenitalis attacking the prepuce and sulcus of the glans penis, the inguino-crural fold and perineal region, is frequent during the later secondary stage of individuals never previously subject to herpes. The undefined ephemeral dermatoses are evanescent but have no subjective symptoms. They present themselves in linear or crescentic formation and remain erythematous, never

(1) Practical Medicine Series, September, 1902.

changing to papules or other progressive lesions. They remain stationary for a very short period subsiding as abruptly and insidiously as they appeared, and leave nothing to indicate their previous existence. Their favorite location is the abdomen. The complementary dermatoses of syphilis are nonsyphilitic in nature and do not differ from similar dermatoses, except in possible severity. They are apt to be protracted and intractable to treatment. Specific treatment is without effect, except at times when it intensifies the lesions.

Autotoxic rashes simulating exanthemata in adults are reported by M. Rosenwasser.[1] The first case presented all the premonitory variola symptoms: headache, extreme backache, pain shooting down the limbs, fever, subsidence of fever on the third day followed by a papulomacular eruption simulating in appearance and distribution beginning variola which did not reach the umbilicated vesiculo-pustular stage. In the other case a seemingly characteristic measles rash occurred with the prodromal malaise and fever but without the eye, nose, throat, and bronchial-mucous membrane symptoms. The symptoms disappeared in each case on removal of autotoxemia. Rosenwasser believes with the older clinicians that the bowels in suspected exanthemata should be emptied before diagnosis is made.

Erythema visceral crises may, as was pointed out last year, be mistaken for appendicitis or intussusception, demanding operation.[2] J. S. Chenoweth reported[3] to the Louisville Medical Society, November, 1896, two cases of appendicitis occurring in connection with erythema exudativum multiforme. The first occurred in a boy of 15, whose mother had rheumatism and tonsillitis. One maternal aunt had rheumatism and another, exudative erythema. The father had rheumatism and died of endocarditis and the sister suffered from erythema nodosum. The boy had been under care for six years for tonsillitis, epistaxis, colic and urticaria. December, 1895, he presented symptoms of appendicitis complicated with erythema, hematuria and epistaxis. On opening the abdomen, the appendix was found to be 3 inches long, distended by

(1) Cleveland Med. Jour., July, 1905.
(2) Practical Medicine Series, September, 1904.
(3) Matthew's Quarterly, January, 1897.

mucus and blood clot. Hemorrhage of the walls of the appendix and mesentery had occurred. The tenth day after operation erythema appeared on the abdomen. During the following weeks, every phase of exudative erythema, pain and edema in the muscles and joints, urticarial lesions of all types, bleeding from the nose, hematemesis, hematuria, and intestinal hemorrhage, occurred always preceded by intense colic. Purpuric spots appeared in the legs. The boy recovered from the operation but for the next decade had colic, erythema and hematuria. In another boy, whose sisters suffered from dysmenorrhea, colic, tonsillitis and urticaria, erythema exudativum with appendicitis occurred. On operation the appendix was found hemorrhagic and containing only a little mucoid material with fecal odor. Chenoweth believes that the pathologic condition of the appendix is similar to that taking place in the skin and the condition produced by the erythema predisposes to appendicitis.[1] The *dermatoses of appendicitis* described by Moty,[2] varied with the type of the intestinal disorder in which they were found. They should be separated from the erythemas as being secondary to the constitutional condition produced by the appendicitis.

Erythema nodosum has as *complications* cardiac disease, hemorrhagic nephritis, neuralgia, peripheral nerve paralysis, multiple neuritis, mania and melancholia, according to I. A. Abt.[3] Most children suffer from anemia after disappearance of the dermatosis. It predisposes to tuberculosis and has been noticed as a complication of scarlatina, measles and malaria. It is liable to occur in cholera, septicemea, rheumatism and scurvy. According to Hoffmann,[4] it is an infection and should not be classed with the multiforme types. He claims it is due to a localized inflammation of the subcutaneous veins arising from invasion of the blood-stream by staphylococcus which probably enters the body through the tonsils. Like Unna, Hoffmann signally fails to establish this staphylococcus by Koch's law.

Borax eruptions, according to Lewin,[5] belong to the ery-

(1) Medical News, March 4, 1905.
(2) Practical Medicine Series, September, 1904.
(3) Amer. Jour. of Derm., January, 1905.
(4) Deutsche méd. Woch., Dec. 15, 1904.
(5) Untoward Effects of Drugs, 1881.

thema group. According to Poulsson,[1] erythema, bullous eczema and psoriasis may be produced by minute quantities of borax taken daily in food in which borax has been employed as a preservative. The employment of borax in epilepsy was abandoned largely because of disagreeable dermic and intestinal untoward effects produced by it.

Eruptions in typhoid are discussed by R. v. Jaksch.[2] In one case a hemorrhagic eruption appeared around the umbilicus toward the end of the fever. It had nothing in common with typhoid roscola, and bacilli could not be found in it. In v. Jaksch's opinion it was dermic manifestation of widespread hemorrhage into the muscles; a view supported by the coexistence of urobilinuria and the patient's subicteric appearance. Another eruption more common, which appears during the first week, consists of rather large papules with a pale summit. The blood from these contains typhoid bacilli.

Pompholyx, according to A. M. Crispin,[3] has its origin in absorption of by-products from the intestinal canal or in faulty hepatic metabolism. It is a skin manifestation of incompletely oxidized products. A vasomotor paralysis results which may terminate in inflammation of the orifices of the sweat apparatus. The sweat having undergone chemical changes irritates the skin on passing through it. The eruption is generally localized on the hands between the interdigital spaces, on the palmar surfaces and sometimes on the extensor surface of the forearm. Next in frequency the foot is affected and it sometimes extends to other parts of the body. The face is never affected. It is most common in adolescents and middle aged, but is not often seen in children or in old age. It is more common with women in England and with men in America. It may assume the aspect of a true eczema. Kaposi claims that it is a true eczema from the beginning. The duration is variable. It may last from a few days to two weeks. It is frequent in spring and autumn and sometimes appears periodically.

Nail disease accompanied by arthritis of distal finger and toe joints is reported by M. B. Hartzell[4] in a 41-year-

(1) Norsk. Mag. f. Laegevid., November, 1904.
(2) Ztschrift. f. Heilkunde, B. XXVI.
(3) Amer. Jour. of Derm., January, 1905.
(4) Univ. of Penn. Med. Bull., 1904, p. 204.

old man. Fifteen years before coming under observation, the tip of the right thumb became red, swollen and painful. The redness and swelling, gradually extended back to the first joint. A month or two later, alterations in the nail occurred. At irregular intervals each of the fingers were later attacked similarly. The nail changes were always preceded by redness and swelling of the finger tips, which gradually involved the entire phalanx. The swelling of the ends of the fingers disappeared after a time but the joints remained thicker than normal. Thirteen years later, the ends of the great toes were similarly but less severely affected. The disease spreading to the joints later. No evidence of onychomycosis, rheumatism, syphilis or dermatoses could be obtained. Under treatment, which consisted in nightly application of mercurial ointment to the nails and nail bed, and internal administration of arsenic in moderate doses, decided improvement resulted in the nails. Skiagraphy showed the finger swellings were due to changes in the soft parts only. The only similar case, Hartzell finds, is one reported by Rist.[1] A 35-year-old woman had nail dystrophy preceded by swelling, redness and pain of the finger and toe tips. The disease began in the right great toe, attacked the others in succession and finally the fingers, lasting four months. The nail first loosened at the free border, then became friable and then fell off, leaving the nail bed bare. After about two months the swelling disappeared, except about the joints. At no time was there ulceration or suppuration. The process was completely dry. The patient suffered from hydrarthrosis of both knees and had trouble with her wrists and ankles.

Pathologic pigment increase, according to Fick[2], is not an etiologic factor in tumor growth. Cell proliferation and pigment formation are coordinated processes. The only nevus cells pigmented are those in close apposition to melanoblasts. Melanoblast processes (chromatophores) lie partly between nevus cells and in the cutis deeper layers about the vessels. The pigmentation of nevus cells is similar to that of epidermic cells. The melanoblasts are very early differentiated from the mesoderm and

(1) Ann. de Derm. et de Syph., VIII.
(2) Jour. of Cut. Dis., January, 1905.

elaborate pigment at the expense of the hemoglobin diffused there. They do not move, but the granules in their protoplasm exhibit Brownian movement. In the course of development they are englobed in the epidermis or in the hair, according to J. C. Jonston. They reproduce themselves indefinitely. Melanoma indicates that melanin is brought to the tumor cells from the outside. Alveoli of tumor cells are often practically pigmentless while surrounded by masses of chromatophores whose branches thrust between them can be traced to a point next the endothelium of capillaries. In the endothelium, fragments of hyalin material reacting like stroma of red cells are englobed in places. In certain protozoa, nuclear chromatin is capable of transformation into pigment. A nevus described by Jonston has nuclei containing a pigment deposit. At full development the nuclear structure was transformed into a mass of brown granules surrounded by a clear membrane.

Regional diagnosis of dermatoses is discussed by R. W. Schoenle,[1] who gives the following table, modified from Pye Smith:

Scalp: Eczema, seborrhea, alopecia, alopecia areata, psoriasis, syphilis, steatoma, favus, ringworm (in children), pediculosis.

Face—Forehead: Chloasma, syphilis, psoriasis, acne, zoster, epithelioma. Eyebrows: Seborrhea, alopecia areata, alopecia syphilitica. Eyelids: Xanthoma, milium, eczema tarsi. Nose: Lupus, epithelioma, rhinoscleroma, rosacea, seborrhea. Nose and Cheeks: Rosacea, lupus erythematosus. Nostril orifice: Folliculitis, impetigo, herpes. Upper lip: Eczema, herpes, lupus. Lower lip: Epithelioma, syphilis. Mucous membrane of mouth: Herpes, syphilis, measles, smallpox, leucoplakia, lupus, lichen planus, pemphigus. Bearded face: Sycosis, pustular eczema.

Ears: Lupus erythematosus, lepra, xanthoma tuberosum, syphilis, eczema.

Neck: Scarlatina, eczema, intertrigo, furuncle, carbuncle, sycosis.

(1) N. W. Medicine, November, 1904.

Back: Acne, tinea versicolor, pediculosis, seborrhea, carbuncle.

Chest: Scarlatina, varicella, syphilis, seborrhea, keloid, lenticular cancer. Breasts: Eczema, keloid.

Nipple: Scabies, eczema, Paget's disease.

Sides of Trunk: Zoster, syphilis.

Abdomen: Typhoid and typhus rashes, tinea versicolor, syphilis, scabies. Umbilicus: Scabies, carcinoma, erysipelas.

Scrotum: Eczema, pruritis, syphilis, elephantiasis.

Prepuce: Scabies, herpes, syphilis, chancroid, eczema.

Nates: Furuncle, carbuncle, scabies, syphilis.

Anus: Eczema, pruritus, mucous tubercles.

Elbows—Flexor side: Eczema, xanthoma planum. Extensor side: Psoriasis, ichthyosis, xanthoma tuberosum.

Forearms and Backs of Hands: Erythema multiforme.

Wrists—Flexor side: Scabies, lichen planus. Extensor side: Smallpox.

Hands and Feet: Eczema, scabies, callosities. Palms and soles: Eczema, syphilis, pompholyx. Fingers and toes: Chilblains, pompholyx. Nails: Hypertrophy, onychomycosis, onychia, paronychia, atrophy.

Axilla and Groins: Eczema, intertrigo, ringworm, erythrasma.

Thighs—Extensor side: Prurigo, keratosis pilaris.

Knees—Extensor side: Psoriasis, ichthyosis. Flexor side: Eczema.

Legs: Eczema, elephantiasis, ulcers, erythema nodosum, purpura, ecthyma.

Universal: Eczema, psoriasis, ichthyosis, erythema scarlatininoides, dermatitis exfoliativa, pityriasis rubra, lichen acuminatus.

Tuberculides in childhood have been examined by Nobl,[1] who found the papulonecrotic tuberculide 13 times among 430 children with various disorders. This is commonly discrete but sometimes grouped. It is usually present on the extensor surfaces of the extremities, lumbar region and buttocks. It is the "folliculitis" of Barthelemy. The children in whom it occurred presented evidences of scrofulotuberculosis most commonly lymphadenitis. Eleven out of

(1) Derm. Zeitschr., XI, Hft. 12.

12 presented scrofulous gummata and lichen scrofulosum also. No tubercle bacilli were found. The condition is probably due rather to dermic irritation by toxins than the weakened bacilli to which Nobl ascribes it.

Jaundice and xanthoma multiplex are usually concomitant. H. H. Whitehouse,[1] however, reports 7 cases (4 in males and 3 in females), in which jaundice was entirely absent. They were all mild cases. If the jaundice be chargeable to xanthoma of the bileducts, then absence of bileduct implication explains the cases.

Acute pemphigus accompanied with delirium is reported by Lesser.[2] There was acute generalized pemphigus with marked rise of temperature and hallucinatory delirium, requiring transfer of the patient to the psychiatric department. Lesser is of opinion that the case was of infectious type. Ziehen, however, claimed that it was of the alcoholic delirium type. In other words, that pemphigus occurring in alcoholism is apt to be followed by delirium. The psychic symptoms are such as readily occur from any toxic or autotoxic cause.

Senile eczema, according to M. Leale,[3] is a dermatosis with initial onset during old age, attributable to senile circulatory changes and resultant degenerations.

Post-exanthematic lupus vulgaris is reported by Von Veress[4] as following varicella. Von Veress is of opinion that lupus following exanthemata is due to external inoculation. The numerous dermic lesions, the absence of internal metastases and of generalized emboli, the appearance of disseminate lupus after the acute exanthem, the frequent increase in the eruption at a later period, all indicate external origin through inoculation; a multiplicity of lesions moreover may result through numerous traumata which exist in the skin of children. Implication of the vessels in the structure of the lupus nodules and the appearance of nodules in all the dermic layers; features necessary to the hematogenous theory, are absent.

Acne and the sexual organs are discussed in relation by Jacquet and Rondeau,[5] who point out that there is a rela-

(1) Boston Med. and Surg. Jour., Dec. 8, 1904.
(2) Berlin klin. Woch., Sept. 5, 1904.
(3) Amer. Med., April 15, 1905.
(4) Monatsheft f. Prakt. Derm., XI, No. 11.
(5) Presse Médicale, March, 1905.

tionship between vernix caseosa, hereditary seborrhea and fetal acne. The vernix, according to their results, is an acute keratoseborrhea produced by a pathogenic cause originating in the genitory plasma, fixed by endocutaneous fetus stimulation. Another factor is the direct influence of parental seborrhea. There is an analogous relation in the fetus between development of the mammary glands and facial acne. The quiescence of the genital system in early childhood, and its awakening at puberty point to adolescence seborrhea being a revivifying embryonic evolution. Toward the end of the fourth month of pregnancy, sexual indifference comes to an end, and henceforth genital system evolution is particularly active. The ovaries and seminiferous ducts are formed. The uterus of the new born hypertrophies and its mucous membrane shows the usual menstrual alterations; some girls have a true menstrual discharge soon after birth. The prostate increases in size, the testicles swell, and at birth hydrocele is sometimes present comparable to that of the adult. During this time the galactiferous ducts swell and their epithelium desquamates. Sometimes the swelling is decidedly marked and there is appreciable secretion of colostrum. A crisis occurs at this intrauterine period similar to that of a miniature puberty which is attended by cutaneous stimulation as in adults. There is marked development of hair, seborrhea in the shape of fetal milium, acne with comedones and the possibility of becoming indurated. Sebaceous milium and fetal acne are signs of a special organic stimulation; concomitants of a miniature puberty. Vernix caseosa is a fatty keratosis related to the pilo-sebaceous function influenced by the same stimulation as the milium itself.[1] The sexual development of puberty, remarks Havelock Ellis,[2] involves hair development in regions previously hairless. As the sebaceous glands are the vestiges of former hairs surviving from the period when the whole body was hairy, they have an abortive tendency to this same impulse. With the development of puberty there is correlated excitement of the whole pilo-sebaceous apparatus. In regions where this is vestigial and notably in the face, this abortive attempt of the hair follicles, and their sebaceous appendages to

(1) Medicine, September, 1905.
(2) Psychology of Sex: Sexual Selection in Man.

produce hairs tends only to disorganization and simple comedones or acne pimples are liable to occur.

For the *production of acne,* according to P. Gaston,[1] there must be two factors: the provocative pathogenic agent and the culture medium or predisposing soil. The second factor is the more important. In order that the pathogenic agent should develop, there must occur modifications of the sebaceous secretion due to elimination by the glands of abnormal products. These modifications can be easily found in certain cases, either in the products of sebaceous excretion or in the general organic secretions. These are the symptomatic acne (occupational or toxic infectious), but in other cases the modifying factors are not clearly discernible and these are the acnes called "primary" or "essential." In reality, according to Gaston, there are no primary acnes, and in disorders of glandular elimination—in particular the urinary—are found explanations of the appearance of seborrheic states. In 6 cases where the urine was carefully examined he found an increase of urea, of acidity and of uric acid so it would appear that acne is accompanied with a suboxidation state which acts on the secretions of the sebaceous glands producing modifications in the quality and quantity of sebum. Gaston has not carried his examination into the domain of the renal products resulting from intestinal fermentation.

The *influence of smallpox on vaccination* is discussed by J. C. Hibbert,[2] who presents the following table of smallpox successfully vaccinated or revaccinated after appearance of the eruption.

The Guarnieri bodies of variola and vaccinia, according to Siegel,[3] are genuine parasites belonging to a new group of protozoa. They are extremely small, can be identified only with the highest microscopic powers and pass through a Chamberland filter. In foot-and-mouth disease a similar protozoon is found which sporulates only in the nuclei of the epidermal cells, while the smallpox one sporulates only in its protoplasm.

Herpes zoster and croupous pneumonia, Riehl[4] found

(1) Arch. gén. de Méd., April 4, 1905.
(2) Lancet, May 20, 1905.
(3) British Med. Jour., Oct. 29, 1905.
(4) Munch. med. Woch., 1904, No. 51.

Age.	Date of primary vaccination; evidence of same.	Date of appearance of smallpox eruption.	Date of vaccination or revaccination performed after appearance of smallpox eruption.	Evidences of successful vaccination or revaccination performed after appearance of smallpox eruption.	Type of disease.
53	Infancy; two fair scars, a quarter square inch in area;	Oct. 17, 1904.	Oct. 17, 1904.	Vesicles at sites of revaccination on Oct. 22.	Discrete.
36	Infancy; one fair scar, three-eighths square inch in area.	Feb. 4, 1904.	Feb. 4, 1905.	Papules at sites of revaccination on Feb. 9.	Discrete.
44	Infancy; one fair scar, one square inch in area.	Dec. 14, 1904.	Dec. 14, 1904.	Well-marked vesicles at sites of revaccination on Dec. 18.	Semi-confluent.
24	Infancy; one fair scar, two-fifths square inch in area.	July 16, 1904.	July 17, 1904.	Vesicles at sites of revaccination on July 22.	Discrete.
23	Unvaccinated.	March 15, 1905.	March 16, 1905.	Well-marked vesicles at sites of revaccination on March 19.	Discrete.
45	Infancy; one good scar, half square inch in area.	March 6, 1905.	March 7, 1905.	Papules at sites of revaccination on March 12.	Discrete.
16	Unvaccinated.	March 5, 1905.	March 7, 1905.	Well-marked vesicles at sites of vaccination on March 11.	Discrete.
15	Unvaccinated.	March 14, 1905.	March 16, 1905.	Well-marked vesicles at sites of vaccination on March 19.	Semi-confluent.
57	Infancy; one faint scar, one-eighth square inch in area.	March 3, 1905.	March 6, 1905.	Papules at sites of revaccination on March 11.	Discrete.
29	Infancy; one good scar, three-quarters square inch in area.	Feb. 20, 1905.	Feb. 23, 1905.	Well-marked vesicles at sites of revaccination on Feb. 27.	Discrete.
32	Infancy; one faint scar, one-eighth square inch in area.	Feb. 21, 1905.	March 6, 1905.	Papules at sites of revaccination on March 12.	Discrete.

coexistent in 129 cases out of 481, or 26.82 per cent. Drasche found it in 40 per cent of the cases; Metzger in 43.2 per cent; Smaler in 32 per cent. Of the 129 cases, 99 were men and 30 women. Herpes is most common in robust patients of 20 to 30 years, and infrequent in the very young and very old. The eruption usually occurred on the third, fourth or fifth day of the disease, followed soon by the crisis. Sometimes it appeared on the second day or was delayed to the ninth or tenth. In one instance it occurred on the twenty-sixth. Recurrence during convalescence as reported by Bleules was not noted. Of the fatal cases, only 6 had an eruption of herpes. The dermatosis is most abundant in mild cases. In 88.36 per cent the eruption occurred in the region of the second or third branches of the trigeminus. The upper lip was involved in 25 cases; upper and lower lip, 24; upper lip and angle of the mouth, 1; upper lip and nose, 7; upper lip, lower lip and nose, 3; upper lip, angle of mouth and nose, 2; upper lip and chin, 1; upper lip, lower lip and chin, 1; lower lip, 16; lower lip and angle of mouth, 4; lower lip and nose, 2; nose and nasal septum, 15; angle of mouth, 8; angle of mouth and chin, 1. Herpes appeared on the same side as the pneumonia 12 times; on the opposite side 12 times. In 46 cases of right-sided and 39 cases of left-sided pneumonia, it occurred on both sides; in 17 cases of double monia, it occurred on both sides; in 17 cases of double pneumonia it appeared thrice on the right side, thrice on the left side and eleven times on both sides. It appeared once on the tip of the tongue, thrice on the ear, twice on the forehead and twice on the upper eyelids.

Leprosy is declining in Norway, according to G. Armauer Hansen,[1] the Inspector General of Leprosy. The two great leper colonies in Norway are Sondfjord and Nordmore. These two districts are similar as far as conditions of life and position are concerned. Both lie on the coast and the inhabitants of both are fisher-folk who live chiefly on fish. Cleanliness, both personal and domestic, was long at a very low level, but the improvement in this respect was first evident after diminution of leprosy had well advanced. Since the decade ending in 1860, the new cases in Nord-

(1) Scottish Med. and Surg. Jour., January, 1905.

more have diminished from 53 to 25 per cent, and in Sondfjord from 28 to 16 per cent. The lepers in Norway are now so few that two of the asylums were closed in 1894, and the oldest of the institutions will be closed when the last of its present patients are dead. The remaining asylums, together accommodate 500 patients. The figures show that leprosy was on the increase in the middle of the 19th century, but soon after began to fall. Supervision and isolation have had much to do with the decrease albeit, at the same there has been an improvement in curing fish, but the decrease began before the improvement in fish, but practically coincident with the improvement in supervision and isolation.

Acanthosis nigricans after breast cancer is reported by Menaham Hodara[1] in a 30-year-old woman in whom the acanthosis had appeared two months after the onset of the cancer. The skin about the mouth and nostrils was dark brown and blackish; thickened and having numerous small furrows and wart-like elevations. There was some pruritus but no scaling. The face improved somewhat after removal of the breast and the disease seemed to be at a standstill. Six months later it rapidly involved the entire face, neck, trunk, axillæ, folds of the elbows and genitals. The operation scars were not pigmented. The patient was in excellent health and there was no evidence of cancer recurrence.

The *bacteriology of chromidrosis* has been studied by R. Trommsdorf[2] in the case of a woman with intensely red perspiration of which she could not rid herself despite the greatest cleanliness. The perspiration was not of bad odor. The cultures contained red and yellow bacteria. According to Trommsdorf red and yellow sweat are due to correspondingly hued bacteria. Different chromogenic microbes are found in any one case. The microbes in the present case were leptothrices and Trichomycosis palmellina. Blue perspiration has been found associated with indican dermic excretion, albeit Trommsdorf inclines to refer it to a bacterial origin.

Copaiba eruptions simulating scarlatina are reported by

(1) Monattshfte f. Prakt. Derm., XI. No. 19.
(2) München. med. Woch., Dec. 11, 1904.

D. McWalter[1] in a case of a gonorrheic young man to whom 20 minims of balsam copaiba were given followed by a rubelloid eruption and a temperature of 102° F. falling next day to 101° F. and followed by tonsil enlargement, congestion and a general erythema. In three days there was marked desquamation, casts of the fingers were thrown off and during this time there was albuminuria. Complete recovery occurred in twelve days.

Halos and "odors of sanctity" of neuropathic origin are discussed by *Medicine*[2] which points out that about a quarter of a century ago W. A. Hammond, in a paper read before the American Neurological Association, called attention to the fact that the odors of sanctity ascribed to the saints by Gorres[3] had a neuropathic explanation. He pointed out that odors were emitted during conditions of hysteric excitement, of sexual excitement, and of religious exaltation, which so often vicarates with these two conditions. Féré[4] has recently described three cases which give a like explanation of the appearance of the halo. In two of these he has witnessed the phenomena. Two of them were cases in migrainous females, in whom, during the course of a migraine crisis, there suddenly appeared around the head an orange-colored light stretching out from it for 8 inches. At the same time the skin had an orange tint. In one of these patients the phenomenon lasted several hours, in the other but a few minutes. The third case was that of a woman subject to being suddenly awakened by subjective sensations with anxiety, in whom the halo accompanied the anxiety. These cases are the only ones on record where halos have appeared in consequence of neuropathic phenomena. They explain certain appearances sworn to in witchcraft trials.

(1) Lancet, Dec. 5, 1904.
(2) Medicine, November, 1905.
(3) Neurologic Contributions, Vol. 1, 1881.
(4) Revue de Médecine, 1905. No. 4.

CHAPTER II.

SPECIAL DERMATOSES.

Pityriasis rubra of Hebra, according to Nicolau,[1] occurs in three groups; one closely related to tuberculosis, the second intimately associated with pseudoleukemia, while the third is a true idiopathic pityriasis rubra.

Palmar eruptions of the type placed under eczema, eczema seborrheicum and syphilis have, according to Stelwagon,[2] *features in common* when the eruption is essentially chronic, of dry scaly character, and practically limited to the palms or palmar aspects.

Acne telangiectodes has been reported by Pick[3] in two cases. There were numerous small nodules distributed over the scalp, face, upper chest, forearms, hand and legs. Part of these suppurated and part were absorbed. The lesions were most abundant on the face and their disappearance left depressed scars like those of acne varioliformis. The course extended over months. The lesions were a granulation tissue containing numerous epitheloid and giant cells. The sections contained no tubercle bacilli. Acne telangiectodes, according to Pick, is not identical with lupus follicularis disseminatus, but is identical with acneitis and must be distinguished from folliculitis. There is no etiologic relation to tuberculosis, it should be separated from the tuberculomata and the tuberculides. It does not belong to acne as it does not originate in the sebaceous gland. Implication of the sweat-glands may have etiologic significance.

Multiple sebaceous cysts. Alexander McPhedran,[4] of Toronto, reports a case occurring in a 25-year-old man which is remarkable for the large number of tumors present. See Plate I. The skin affection was first noticed dur-

(1) Annales de Derm. et de Syph., 1904, No. 9.
(2) Jour. of Cut. Dis., January, 1905.
(3) Arch. f. Derm. u. Syph., LXXII.
(4) Jour. Cutaneous Diseases, March, 1905.

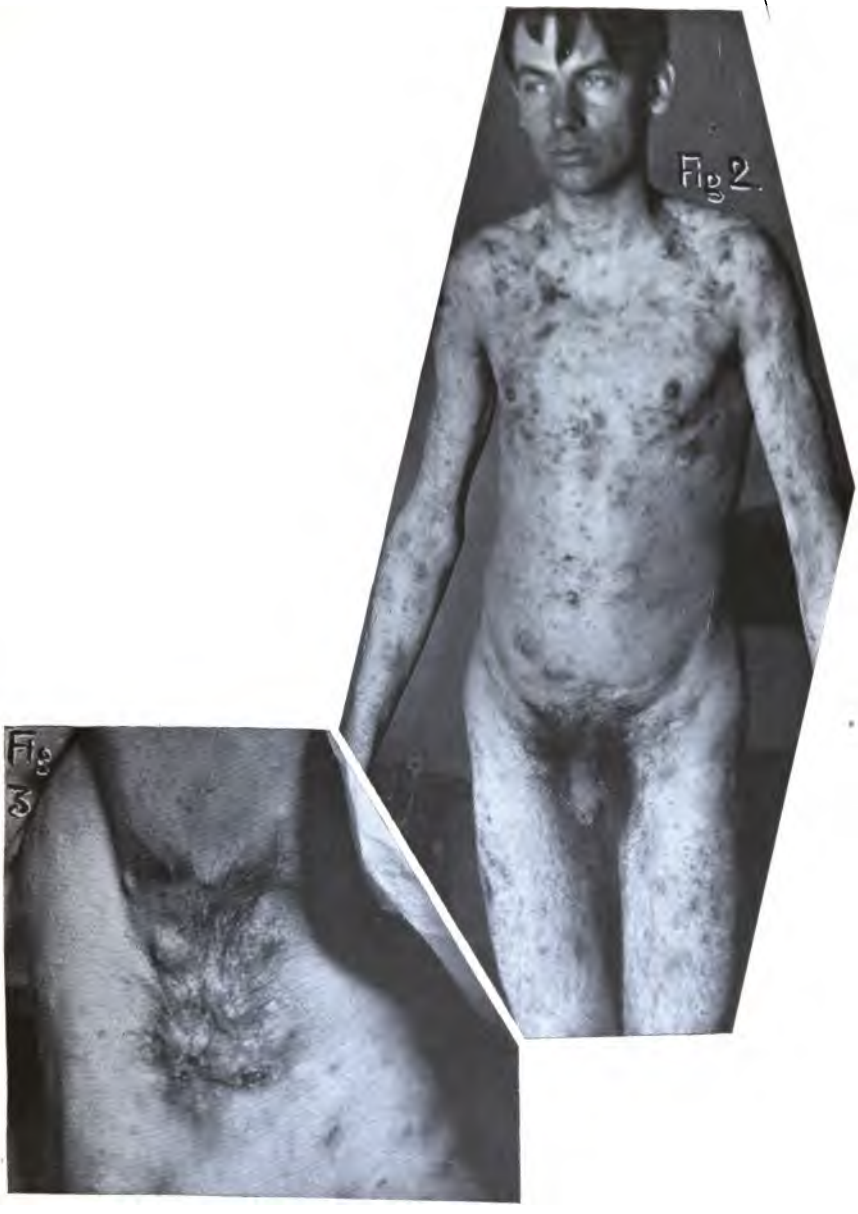

Plate I

ing adolescence and first attracted attention through the occurrence of acne and the formation of large pustules. The lesions could be felt as nodules beneath the skin varying in size, the smallest being barely palpable, the largest fully two centimeters in diameter. On the body they were so numerous that the point of the finger could scarcely be placed anywhere without touching one or more. The small nodules were as a rule deeply placed, and only attached to the superadjacent skin by an ill-defined strand of fibrous tissue, doubtless the obliterated duct. The contents of the smaller and some of the larger nodules consisted of thick, sebaceous material that exuded in a white, ribbon-like form through the linear puncture made by the bistoury. In some of the larger nodules the contents were partly sebaceous and partly a yellow oil. None of the cysts were pedunculated, but as they grew larger and older some of them became inflamed. The wall of the bleb usually sloughed, leaving a large, ulcerated surface, which healed with a broad deep scar.

Folliculitis of the skin and conjunctiva is reported by H. G. Anthony.[1] The skin lesions appeared on the wrist in successive crops as well as on the ears, elbows, nose and thighs. Some terminated without scarring, but most pitted. There were no scars or lesion relics on either eye. The eye lesion when first observed presented the appearance of phlyctenular conjunctivitis, but after four months it assumed the characteristics of episcleritis. It consisted of two small nodules at the outer corneoscleral margin of the left eye, having blood vessels running into it. The skin lesions became an acuminate papule, within twenty-four hours, first red, then blue, in color. Some capped with a pustule, later drying, with an adherent crust, and necrotic plug. Exfoliation of this left a varicelliform scar. Some papules underwent involution in from ten to twenty days without scar. With some crops appeared a few bean-sized, deeply situated lesions, which did not come to the surface, and required three months for absorption.

Lichen spinulosus, according to H. G. Adamson,[2] is a dermatosis occurring in children, generally boys, characterized by formation of fine filiform spines in groups dis-

(1) Jour. of Cut. Dis., August, 1905.
(2) British Jour. of Derm., March, 1905.

tributed over the trunk and limbs. These spines arise from the pilosebaceous follicles, the mouths of which are slightly raised and form pinhead-sized papules either normally colored or slightly red. There is no itching and little if any disturbance of general health. The process is essentially a hyperkeratosis of the follicle; perifollicular inflammation being slight or absent.

Under the title of *universal congenital atrichia,* A. A. Eshner[1] reports a case where there was total absence of hair which condition had been congenital. The skin was soft, smooth and unctuous. The man perspired but little. The finger and toe nails were only about half the normal length, their distal extremities were irregular and longitudinally rugose. The coexistence of imperfect teeth and hairlessness was pointed out in the present series about three years ago.[2]

A polymorphic purpuric *erythema following Pasteurization of rabies* is reported by Nicolas and Favre[3] in an 8-year-old girl, who was bitten by a dog and given antirabic vaccination for 18 days, during which her health remained excellent. Ten days after conclusion of treatment, the girl was returned to the Pasteur institute, because of general malaise, headache, pain on eating, slight diarrhea and pain at the waist. With these appeared the eruption described—which was chiefly seated at the right side in the center of the left haunch, around the two knees and on the posterior aspect of the two elbows. There was slight albuminura which disappeared in about two days and the general health soon became excellent. The eruption completely disappeared later.

Dermatitis from jasmine frictions is reported by F. Koren.[4] At the close of several frictions with oil of jasmine, made for gout the patient was attacked by a generalized papulovesicular dermatitis, especially marked at places where the clothing pressed. There were some red patches in the mouth and there was dysphagia. The eruption was followed by complete desquamation resembling that which follows scarlatina. There was well marked fever, particularly at the time when the exanthem retroceded. While

(1) Amer. Jour. of Med. Sciences, April, 1905.
(2) Vol. 10, 1902.
(3) Lyon méd., Jan. 29, 1905.
(4) Norsk. Mag. for Laegevid, 1905, No. 1,

jasmine taken internally has produced dermatitis quite frequently, eruptions from its external use are rare, according to Rasch.

A case of *dermatitis exfoliativa neonatorum* is reported by A. G. Patek[1] who states the disorder is liable to be confounded with pemphigus foliaceus, acute pemphigus and syphilis. While the dermatosis occurs in epidemic form in foundling asylums, according to Ritter, who first described it, it is probably not contagious. Patek found diplococci in its serum. A similar diplococcus found in pemphigus resembles the gonococcus.

Scabies, according to J. N. Hyde,[2] varies to some extent with the presence of immigration and popular movements. Hyde does not take into account the fact that scabies was at one time nearly endemic in the Northern states and in Scotland, especially in the Highlands. Its prevalence was to some extent due to the belief that it was a constitutional disease; a belief in which the famous Psora doctrine of Hahnemann had its origin, traces of which still survive in the term "psoriasis." Hahnemann denounced the doctrine of the parasitic origin of scabies with great fierceness. The doctrine was established even among the regular profession with considerable difficulties. While personal cleanliness did much to wipe out scabies, Hahnemannism tended to prevent scabies being destroyed, as a parasitic disease. Psorinum or the nosode from itch pus is still used in homeopathy on the indications given by Hahnemann and was the subject of an animated discussion before the International Homeopathic Congress of 1893.[3] The prejudice against the parasitic origin of disease in the early 19th century, arose from the prejudices which Hahnemann voiced.

Scabies, according to E. W. Ruggies,[4] *is increasing of late years,* but is not frequently recognized as the typical characteristics are then absent. The pruritus is out of all proportion to the lesions, consisting of isolated scratch marks, small papules and occasionally minute pustules. Some burrows, excoriations, papules or vesicles are found between the fingers and toes. The acarus is difficult to iso-

(1) Jour. of Cut. Dis., June, 1904.
(2) Amer. Jour. Med. Sciences, March, 1905.
(3) Medical Current, 1893.
(4) Buffalo Med. Jour., December, 1904.

late, as it is commonly contained in a drop of blood or the dry secretions.

Rhinoscleroma, according to Topplitz, has been[1] seen in emigrants in New York. The bacterium that causes the disease has a very low grade virulence and produces its effect only in persons long associated with sufferers from the disease. It is of long duration and usually incurable. The slower the development of the symptoms, the more favorable the prognosis. It sometimes disappears spontaneously after long continued severe fevers.

Genital scars, according to Arthur Cooper,[2] are more common and more marked in hospitals than in private cases. In many cases of nervous or other low forms of internal syphilis, the genital region is free from scars; a single scar on the skin of the penis suggests syphilis as a rule, occasionally the local chancre; multiple scars on the mucous surface of the penis only suggests the local chancre; multiple scars on both the mucous membrane and skin also suggest the local chancre; inguinal scars, together with scars on the penis suggest the local chancre; an inguinal scar without any penis scar, if venereal at all, suggests gonorrhea; extensive scarring of the penis or groin, or both, suggests phagedena; genital scars with scars on other parts of the body suggest an ulcerating syphilide.

Fat excretion from *sebaceous glands* occurs physiologically, according to Buchke,[3] under favorable conditions.

Paradoxic perspiration has been reported by Zappert[4] in a child. It occurs only on exposure to cold and yields to the influence of heat. In Zappert's case it was confined to the skin of the dorsum of the upper extremities, of the back, of the thorax. The palm, axillæ, forehead and lower part of the body were not affected. The condition occurred during the winter months. He believes that the condition indicates the existence of a spinal sweat-center.

(1) Med. News, April 1, 1905.
(2) British Med. Jour., Jan. 21, 1905.
(3) Berlin klin. Woch., March 20, 1905.
(4) Munch. med. Woch, 1904, No. 45.

CHAPTER III.

THERAPY OF THE DERMATOSES.

Cutaneous infections in infants should be treated, accordcording to D. A. Stros,[1] as follows: The lesion should be first sterilized so as to prevent further infection. This is best accomplished by means of a bath of 1-15,000 mercury bichlorid, or an application of soap and water followed by naphtholated vaselin (1-10). Irritating applications are to be guarded against, lest they lessen the resisting power of the healthy skin and lead to a spread of the infection. Consequently the daily bath should be of simple boiled water, with the addition at times of a little sodium bicarbonate or boric acid. The dressings to be preferred are of simple sterile gauze. Zinc ointment combined with small quantities of boric or salicylic acid makes the best moist application, while the most useful powder is a mixture of zinc oxid, talc and bismuth subnitrate. Certain therapeutic applications, such as solutions of zinc or copper sulphate, are useful in impetigo, while in purulent conditions hydrogen peroxid is an acceptable antiseptic. Iodoform is powerful in this connection, but has disadvantages. Its use should be reserved for certain forms of ecthyma and cutaneous gangrene. In furunculosis, after proper washing with antiseptics and smearing of the surrounding parts with 10 per cent boric acid in vaselin, the lesions should be incised, drained, and dressed with dry gauze. General treatment consists in proper feeding and hygiene, fresh country or sea air, tonics, and in marked cachexia subcutaneous injections of normal salt solution.

Urticaria in children should be treated as follows, according to Dauchez:[2] The child should be given during two or three successive days, two teaspoonfuls of the following mixture as a laxative:

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(1) Jour. de méd. de Paris, 1904, No. 23.
(2) Jour. med. de Brux., March 9, 1905.

Magnesium sulphate
Sulphur (sublimated)
Potassium bitartrate
White honey of each: gr. v.

The child should be restricted to a milk diet and fish, and fruit especially if fresh omitted. The child should be immersed in a gelatin bath (100 to 200 grs.) for about half an hour. Wiping should not be done, and two hours later, preferably in the evening, the following should be applied by a camel's hair pencil:

Chloroform .....gr. iv
Tincture aconit .....gr. vi
Oil sweet almonds .....gr. xc

M. and stir well.

If there should be irritation at night, apply warm solutions of opium or decoction of tobacco, followed by the free use of the following:

Talc
Zinc oxid
Starch equal parts.

An alkaline water should be given during the treatment pure or mixed with milk. As urticaria, like most nervous disorders, has a tendency to return, laxatives should be continued each week. Preserved pork, shell-fish, salt fish and berries prohibited.

The x-ray in recurrent pyrogenic onychitis has given good results according to G. E. Pfahler.[1] A fifty-year-old woman at the age of 16 ran a needle into the left thumb which was followed by a "run-around." At 30, the nail matrix of the same thumb inflamed without apparent cause and healed up under local applications. Since then, four attacks have occurred affecting the same nail and followed by its regrowth. They all followed general debility marked by gastric neurasthenia. The fourth attack seemingly yielded to internal medication which prescribed by the same physician was without effect in the fifth. This continued four years until after x-ray treatment. Potassium iodid and mercury were given internally and corrosive

(1) Jour. of Cut. Dis., August, 1905.

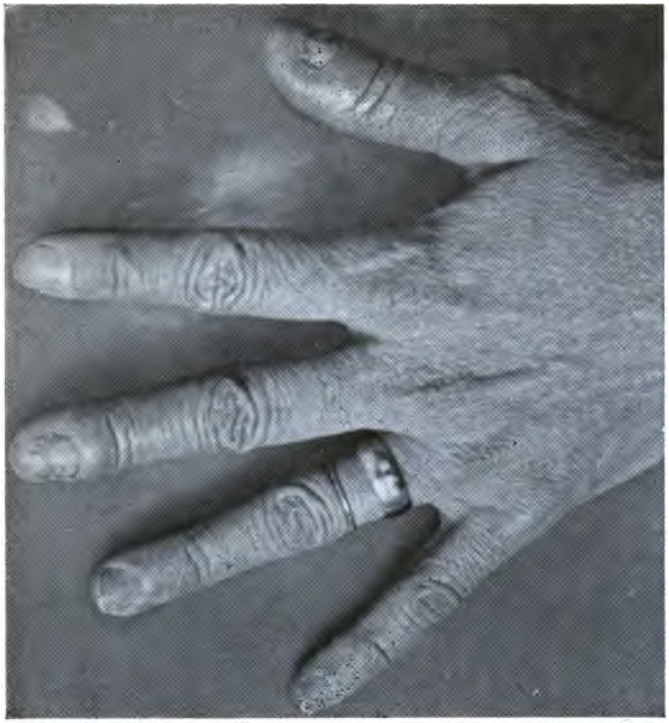

Plate II

Plate III

sublimate used locally without effect for six weeks. All medication was then discontinued and x-ray treatment begun. The thumb and second finger matrix were then deep red in color, indurated and tender as far as the second joint. Under 25 treatments, given for eight weeks (thrice weekly) with the tube six inches distant, a two-and-a-half-inch vacuum five minutes, exposure to one milliampere (Roentgen Ammeter), the condition disappeared. See Plates II and III.

In *senile eczema,* according to M. Leale,[1] the circulation should be properly maintained and elimination carefully secured. Water should be taken frequently in small quantities at a time, so as to secure constant flushing rather than overdistension of the heart and blood vessels. Exercise in moderation is useful. Local treatment should stimulate and thereby improve peripheral circulation in the blood vessels and lymphatics. Carefully regulated and systematic rubbing and frictions best meet these indications. Massage should consist of *effleurage* and *massage a friction.* A small amount of fine olive oil is employed after massage to which is added a pure finely powdered zinc oxid. Each application should be made from 20 to 40 minutes before retiring. Where greater stimulation is necessary, a strong tincture of pix liquida is employed. The patient is given a bath at 94° F., and washed with pure olive oil soap. In most cases recovery occurs in from two to eight weeks.

Disinfectol has been found of value by Shimoyama[2] in *dermatoses of animal or vegetable parasitic origin.* A 50 per cent watery solution painted on the affected parts is said to cure scabies in 48 hours. Disinfectol is a by-product in the fabrication of camphor. It is not chemically pure and the proportions of camphor, terpen, naphtalin, thymol, saffrol, kresol, resin, etc., vary. An eczema sometimes results from its use.

Multiple warts have been removed by Mantelin[3] by the following treatment: They occurred in the case of an 11-year-old girl. She had for three years numerous warts on the lips and hands which were completely removed in

(1) Amer. Medicine, April 15, 1905.
(2) Japan. Zeit. f. Derm. u. G. Ur., 1904.
(3) Jour. de Med. de Paris, April 25, 1905.

one month by 10 grains of magnesia, daily, and the following local application:

℞—Chloral hydrate .....90 gr.
Acetic acid .....90 min.
Salicylic acid ..... 1 dr.
Ether ..... 1 fl. dr.
Collodion ..... 4 fl. dr.

A plantar papilloma is reported by T. D. Berry[1] which was rebellious to all treatment. It was finally removed by the Paquelin cautery, burning entirely through the sole skin into the fatty cushion beneath, and then turning the point round and round, until a hole was left into which the blunt end of a lead pencil could be inserted. There had been no recurrence six months later.

In *acne* the following external application is recommended by J. V. Shoemaker.[2]

℞—Acid salicylic
Resorcin
Phenol *aa*, gr. x.
Ungt. aquæ ros.
Lanolin, *aa*. ziv.
M. ut ft. ung. S. Apply once daily.

Acne treatment, according to G. T. Jackson,[3] should be conducted as follows: Improve the condition of the skin so it will not serve as a culture medium for the bacillus. Empty the skin follicles of bacilli by the curette, acne lancet or the comedo-expressor. Keep the skin constantly aseptic so new infection will not be possible. The patient's general health should be kept up by baths, diet, exercise, proper hygiene and tonics. The best local application is sulphur in the form of the old white lotion.

℞—Zinc sulphate
Potassium sulphuret, *aa.*, gra. 60-120
Aq. rosæ q. s. ad ℥ xxxii.

This is to be shaken before using. Resorcin and sulphur soap are also useful. The x-ray should not be used except in intractable cases, and then only with great care.

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(1) Jour. of Cut. Dis., December, 1904.
(2) Med. Bull., October, 1905.
(3) Med. Record, March 18, 1905.

Freezing in the dermatoses is advocated by A. Julius Berg,[1] who has employed liquid carbonic acid. The action is rapid and intense. In a half a minute marked anemic results followed, in a few moments succeeded by hyperemia and serous exudation. In twelve hours inflammatory redness occurs and small superficial blisters form. These changes disappear in a few days. More intense freezing will produce ulcers. The application is employed three times in three to four weeks. In acne several applications followed by warm alcoholic dressings in the intervals have given excellent results. Psoriasis and lupus, as a rule, are made worse by this treatment. In lupus erythematous the method is sometimes successful.

In *frost bite* Joseph[2] recommends resorcin, alcohol compresses and a 10 per cent calcium chlorid ointment. Euresol (a liquid monacetyl derivative of resorcin) is better than resorcin. He applies it locally as a soap inunction.

℞—Euresol
O. C. terebinth
Eucalyptol *aa*, grs. xxxii
Lanolin
Ung. sapon *aa*. ℥ viii

In *scabies* two essentials are to be met, according to E. W. Ruggles.[3] First, to kill the parasites, not only in the skin, but in the clothing and bedding. The case should be followed until it is cured. Sherwell's washed sulphur treatment is an efficient cleanly method. The patient is rubbed thoroughly with dry washed sulphur powder after a prolonged hot bath and in addition, the sulphur is placed on the lower sheet. Kaposi's ointment:

Naphthol	25 parts
Green soap	50 parts
Prepared chalk	10 parts
Benzoated lard	100 parts

and Stelwagon's ointment:

Sublimed sulphur
Balsam Peru, of each 16 parts

(1) British Med. Jour., Aug. 19, 1905.
(2) Derm. Zentralblatt, 1905, No. 6.
(3) Buffalo Med. Jour., December, 1904.

Naphthol, 1 to 2 parts
Benzoated lard or petroleum ointment, enough to make 64 parts

have given the best results. Two or three applications kill the parasites, but the coexisting eczema is often aggravated by treatment and recovery from it is protracted.

Eudermol (Nicotin salicylate) in scabies has been found preferable, according to W. F. Demitch,[1] to Wilkinson ointment. An ointment of eudermol (1 to 3 grains to the ounce of pertolatum) is a reliable application in scabies. Demitch has not observed any untoward effects.

Uranium plaster treatment of lupus is urged by Norman Walker[2] after the following manner: Uranium oxid is first prepared from the nitrate or other readily obtainable salt, and incorporated with a negative base, like resin or beeswax. The plaster leather having been prepared, a layer of the plaster is applied in the usual thickness to any desired extent. This is quickly covered with waxed paper gummed at the edges to enclose it completely. The plaster is applied at night and removed in the morning. The plasters may be used for months without losing their activity.

Treatment of Psoriasis: Drew[3] advises the following ointment:

℞—Salicylic acid, 150 grains
Chrysarobin
Oil rusci of each, 300 grains.
Green soap
Petrolatum of each, 330 grains.

This is applied from four to six days to the affected parts with stiff brush. On the fifth or sixth day hot baths are given daily, followed by rubbing in petrolatum. This may be repeated several times if necessary, but the patches disappear rapidly after the first treatment, as a rule. The ointment limits the irritating effect of the chrysarobin to the affected area.

In *treatment of pityriasis versicolor,* Gallois[4] recommends a mixture of 8 ounces of hydrogen peroxid and a drachm

(1) Russki Vratch, Jan. 28, 1905.
(2) Scottish Med. and Surg. Jour., September, 1904.
(3) München. med. Woch., Dec. 25, 1904.
(4) Medicine, November, 1905.

of borax. Daily applications remove pityriasis in about two weeks.

Mangrove bark in leprosy is claimed to have given good results by Duque and A. Mareno[1] whose claims are somewhat supported by results at the Havana leper hospital. They claim that in all cases in the early stage, recovery resulted; about half the cases in the second stage recovered. Recovery resulted in only one case in the third stage, albeit marked improvement followed the use of mangrove in many cases. The preparation used was a fluid and a solid extract of the bark of trees of not more than six months' growth. The initial dose of the fluid extract was a teaspoonful night and morning gradually increased until twelve teaspoonfuls were taken daily. The extract was given in pills; 120 grains being the maximum. Local applications of a 30 per cent solution of the fluid extract were also made with baths at night containing enough decoction of the bark to color the water red. In one of the third-stage hospital cases recovery resulted with complete disappearance of Hansen's bacillus.

(1) Le Semaine Med., 1905, No. XL.

CHAPTER IV.

GONORRHEA AND CHANCROID.

The *penetrating power of the organic silver salts* has not been supported by investigations of their action in gonorrhea. As the Editor[1] pointed out in a paper read before the Chicago Academy of Medicine, solutions of the silver salts, organic or inorganic, do not penetrate the mucosa to any great extent. Prolonged pressure of any fluid upon the urethral mucosa causes temporary compression of the capillaries. Removal of this pressure is followed by dilatation of these capillaries, resulting in increased leucocytosis through the walls of the vessel, whose point of least resistance is the lumen of the urethra. This induces more rapid exfoliation of mucous membrane cells and literally carries out the infectious agent upon the free surface. Upon gonococci thus forced into the surface layer of the mucous membrane the silver salts exercise their bactericidal action. The living cell, according to L. Weiss,[2] will absorb crystalloids like potassium, but is impermeable to colloid substances of which the silver salts are made. The fact that these preparations are not precipitated by normal tissue fluids like silver nitrate is, does not give them superior penetrative power. Neither of these two biochemic conditions secures their absorption by the living membrane. Penetration in the living membrane occurs by absorption; in the dead tissue through imbibation.

In the gonorrheal stage of inflammation two types are encountered. In one there is intense ardor-urinæ, lymphangitis along the penile dorsum, blood-stained urethral discharge and meatus and prepuce edema. In such cases, according to H. M. Christian,[3] local urethral treatment should be deferred until the inflammatory symptoms have subsided, which usually takes about ten days. The

(1) Med. News, Oct. 15, 1905.
(2) Ibid., Vol. LXXXV, No. 12.
(3) Medicine, January, 1905.

only local treatment should be immersion of the penis in hot boric acid solution for 15 minutes three or four times daily. For the ardor urinæ and chordee 20 grains of potassium bicarbonate and sodium bromid should be given four times a day. The alkaline mineral waters should be drunk freely. This severe type is not usual. The patient, as a rule, is ready for local treatment when first seen. In the early stage two solutions are ordered—one of potassium permanganate, 1:8000; the other a solution of a silver salt. The patient is instructed to wash out the anterior urethra gently with an ordinary hand syringe full of permanganate solution, repeating the process six times. After this, one syringe full of the silver solution is injected into the urethra and held for ten minutes. This treatment should be employed thrice daily for the first four days, when the strength of the permanganate solution should be increased to 1:4000. About the end of the second week the permanganate solution should be discontinued and the silver solution given night and morning, together with twice during the day, one syringe full of the following:

℞—Bismuth subcarb ..... 2 dr.
Colorless hydrastis ..... 4 dr.
Boro-glyceride .....2 dr.
Distilled water enough to make..32 dr.

Copaiba and sandal-wood should now be given internally. At the end of the third week the silver compound should be supplemented by the employment during the day of an astringent injection. The following is recommended:

℞—Zinc sulphate.
Pulverized alum of each, 12 grains.
Colorless hydrastis, 4 drachms.
Water, enough to make 4 ounces.

Silver iodid injections into the urethra, according to E. H. Siter and A. A. Uhle,[1] cause no burning or irritation if suspended in 5 per cent solution of quince-seed mucilage.

Lactic acid in cervical gonorrhea has been found of great value by S. Chandler.[2] He advises the following procedure: First cleanse the vagina and cervix thoroughly with

(1) Univ. Pa. Med. Bull., January, 1905.
(2) Jour. Amer. Med. Assoc., Oct. 7, 1905.

warm water and cotton soaked in an aqueous solution (4 ounces to 6 ounces) of pyroligneous acid. Expose the cervix by drawing it downward and into view by an ordinary long tenaculum. Then take a hypodermic syringe loaded with pure lactic acid and inject just beneath the membrane a few drops of the acid. Continue this until the whole of the cervix is exposed as the superior and inferior lip is injected. This may be done in one sitting or a nervous case may require two or three.

Sodium chlorid after silver nitrate is, according to W. R. Gries,[1] of great value. The reasons for its use are: The silver salt can be used with greater comfort to the patient as excess silver is precipitated without hurtful results. The effect desired from silver nitrate is accomplished quickly; an excess should not remain in the urethra, as it causes unnecessary pain and does harm. Caustic effect of the silver is eliminated to a great extent. The stronger solutions of silver which are often needed can be used without fear of overaction or caustic effect, since the caustic effect is eliminated to a great extent. The application can be made more general since the influence of excess on healthy tissue is not to be feared. The method of application yields excellent results. The solution of sodium chlorid should be the same as when the silver is injected by a deep urethra syringe. Enough time should be allowed to elapse after application to permit removal of the syringe, to wash it out and to proceed as with the first injection.

Sodium salicylate[2] *in gonorrheal epididymo-orchitis* has been found of value by Carey in doses of 1 to ½ drachms daily in solution. After a day or two of the treatment the symptoms improve rapidly. The pain disappears, as well as the pyrexia and the insomnia. The tenderness likewise disappears, the general condition improves, the testis and cord become of normal size and recovery usually occurs in a week. Induration of the epididymis may persist but yields to potassium iodid. Local treatment is indicated with the salicylate.

Of twenty-six cases of *gonorrheal phlebitis* studied by Heller,[3] twenty occurred in men and six in women. One

(1) Therap. Gaz., Nov. 15, 1904.
(2) Rev. Francaise de Méd. et de Chir., 1904, No. 12.
(3) Berlin klin. Woch., 1904, No. 27.

doubtful case occurred at 34 and the remainder previous to 30. The phlebitis developed as a rule during a first attack. It occurred as early as fourteen days and as late as ninety after infection. The average in fifteen cases was thirty-two days. There were a few cases without other complication. Prostatitis and marked urethrocystitis occurred in one case; epididymitis in six; pleurisy, prostatitis and pyelitis in one; erythema nodosum in one, and gonorrheal arthritis in fifteen. There were varicosities in one case precedent to the gonorrhea. Phlebitis affected the common iliac vein in one case; the internal iliac in one; the femoral in six; the popliteal in one; the deep veins of the leg in one; the internal saphenous in sixteen; superficial veins of the abdomen in three; vaginal veins in one; dorsalis penis vein in one; corpora cavernosa veins in three; pampiniform plexus in one; prostate and bladder veins in two; upper arm veins in four, and forearm veins in one. The veins affected were equally divided on the sides of the body. As a rule the disorder was limited to branches of one venous system, but in six cases two or more widely separated veins were involved. The disorder usually makes its onset with acute pain, followed by firm swelling over the inflammation and doughy edema over the distal distribution of the vein affected. In some instances this edema masked the diagnosis. Fever was present in ten cases; in four it rose above 104° F. Two of the patients had chills. In a few days the swelling subsided and the vein could be felt as a tender indurated cord. Pigmentation, discoloration, ecchymoses and even skin gangrene may follow inflammation of superficial veins. Recovery occurs, as a rule, within a few days to six weeks. In a few cases the vein was permanently obliterated. One case required amputation at the thigh, one patient died of pulmonary embolism, and another of sepsis.

Under *urethritis externa* Moller[1] discusses gonorrheal inflammation of the paraurethral and preputial crypts or passages. The histologic findings are similar to those of excised Morgagni crypts of the urethra. These anomalies are described as paraurethral passages, accessory passages in hypospadias, cutaneous inversions or crypts, atypical

(1) Amer. Jour. of Derm., April, 1905.

sebaceous glands and rapheal passages. Moller treats gonorrheal inflammation by complete excision; primary union generally occurs.

Staphylococcic urethritis is reported by Vannod.[1] The non-gonorrheic forms of urethritis may be caused by different bacteria, such as the streptococcus, diplococcus, streptococcus, bacillus coli and diphtheria bacillus. It has occurred in course of different diseases like typhus, rheumatism, lead poisoning, etc. Tuberculous urethritis has long been known. There are but three cases of staphylococcus urethritis in the literature, all resultant on buccal coitus. Vannod reports the case of a man infected by a woman who had a severe vaginitis. On the fourth day after coitus free purulent discharge occurred with severe burning pain or urination. There was a large quantity of very long thick pellicles in the urine. On the microscopic examination and culture upon agar, pure cultures of staphylococci were obtained. The patient was treated by washing out the bladder and urethra by Janet's method, and complete recovery resulted in fourteen days. Staphylococcus urethritis is very amenable to treatment.

General gonorrheal infection is reported by Prochaska[2] in six cases. In one there was painful knee joint disorder and an eruption simulating erythema nodosum. In three cases endocarditis and polyarthritis occurred. Purulent meningitis appeared in one case and in two there was severe sepsis without localization.

Epididymitis Gonorrheica, according to Oppenheim,[3] has besides violent muscular exertion, as causes mechanic or chemic irritation of the posterior urethra and semen ejaculation. In many cases the vas deferens is not involved, but the inflammation is localized in the tail of the epididymis, later affecting the other parts and the vas deferens.

Gonorrheal pelvic peritonitis, according to Anspach,[4] bears a close resemblance to appendicitis. He gives the following differential table:

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(1) Correspondenz-Blt. f. Schweiz. Aerzte, Sept. 1, 1905.
(2) Deutsch. Arch. f. klin. Med., XLIV.
(3) München. med. Woch., July 22, 1905.
(4) Proc. Philadelphia Co. Med. Soc., Dec. 31, 1904.

APPENDICITIS.

History—Previous similar attacks, habitual overeating, chronic intestinal indigestion.

Onset — After indiscretions in diet.

Pain beginning in epigastrium; not well localized at first; later localized to appendical region.

Gastro-intestinal symptoms — Nausea, vomiting, constipation, etc., more marked.

GONOCOCCUS PELVIC PERITONITIS.

History — Leucorrhea or vesical irritability soon after marriage or suspicious intercourse.

Onset—At or directly after a menstrual period.

Pain in lower abdomen, worse perhaps on one or other side.

Gastro-intestinal symptoms — Nausea, vomiting, constipation less marked.

Eyelid chancroid, according to M. L. Foster,[1] existed in a case where the lid was red, swollen and drooping. There was an ulcer 6 mm. by 2 mm. in the middle of the intermarginal space. The floor was excavated and covered with purulent detritus. The edges were abrupt and elevated but not indurated. No luetic symptoms appeared during the following months. A month before the chaneroid the patient had a penis ulcer and suppurative inguinal buboes.

(1) Manhattan Eye Hospital Reports, March, 1905.

CHAPTER V.

SYPHILIS.

The curability of syphilis and its tests are among the most prominent subjects discussed during the year. The tests of curability are more extended and the present optimism is better based than most of the previous claims.

Some thirty years ago Zeissl, of Vienna, advanced certain decidedly pessimistic opinions as to curability of syphilis which contrast very markedly with the optimistic views recently collated by Aronstam.[2] "Some think," remarked Zeissl,[1] "that when a patient has for some time enjoyed immunity from manifestations of syphilis that he is cured; but I tell you, gentlemen, that if a man contracts syphilis he will die syphilitic and that at the day of judgment his ghost will have syphilis."

Aronstam concludes from a summary of the opinions of leading syphilographers in the United States and abroad that: Syphilis is curable provided treatment be begun early and faithfully carried out. At least three, if not four, years of persistent adequate treatment are needed to bring the morbid process under control with all danger of recrudescence eliminated. Available indications of total eradication are absence of all manifestations for at least two years, a steady gain in bodily weight, or at least no perceptible loss of same, and propagation of healthy offspring. There are a number of elements of error in these last indications. The character of increase of bodily weight should be more clearly put, since syphilis may cause suboxidation states like lipomatosis.

Degenerate offspring may be healthy, yet, as E. Fournier[3] points out, these may result from congenital syphilis. Indeed, as J. G. Kiernan[4] has shown, the old man appearance of the syphilitic child is the result of an arrested development of the senile or simian intrauterine period.

(1) British Med. Jour., June 21, 1875.
(2) Amer. Jour. of Derm., March, 1905.
(3) Heredo-syphilis.
(4) Medicine, 1901.

Furthermore, parasyphiloses often occur long after disappearance of all luetic manifestation. Syphilis in these creates a predisposition which on the application of a proper exciting cause produces non-specific effects. While this predisposition is exaggerated both by syphilographers and alienists as to extent and potency, still it should be taken into account in dealing with the curability of syphilis so far as therapeutic persistency is concerned. The recrudescence of syphilis has received much less attention from a therapeutic standpoint than its importance merits. The problem of recrudescence, as the *Medical Record*[1] recently remarked, still demands careful study, since, despite decades of study, it remains considerable of a mystery. Lesser[2] adopts the older hypothesis of Lang that at the time of infection the virus is carried by the blood into all the tissues and occasions the early manifestations. The virulence of the deposited contagion gradually abates, but it for a time retains ability to incite recrudescences. After a variable period it is either partially or entirely destroyed, and in the former case is modified to such a degree that it loses its infectious tendency. During this dormant period it remains quiescent unless some, often external, stimulus provokes it into action and gives rise to the late manifestations of the disease. This view is in harmony with the clinical observation that the early lesions are apt to be symmetric, while the late are asymmetric. Lesser cites several cases in which recurrences of the skin eruption took place in the pigmented spots remaining from the first exanthem. In the eruptive stage the entire body is flooded with syphilitic poison and that this is deposited more or less uniformly in numerous foci, which serve as a source of relapse during the course of the disease. As the length of time after infection increases more and more of these persisting germs perish and they may finally disappear completely. The practical bearing of this is on therapeusis. In order to hasten diminution and neutralization of these depots of latent virus, frequent repetitions of the course of medication are necessary. Intermittent mercurial treatment has a potent support in this pathogenesis of late syphilitic manifestations. The similar recrudescences in

(1) Jan. 25, 1905.
(2) Senator Festschrift.

malaria suggest that the factor in syphilis is, as in malaria, a protozöon rather than a microbe. At the same time diagnoses between non-specific seeming recrudescences and specific should be made. This is not always done because of the tendency to regard all pathologic phenomena in syphilitics as specific. The latest alleged pathogenic organism of syphilis is a spirillum; the spirocheta. This has been found coexistent with nearly all the types of syphilis in man and anthropoids, but has not as yet complied with Koch's law. Indeed, so far no attempt has been made to test it from this standpoint, albeit luetic inoculation in animals continues to be made. The burden of proof therefore still rests on the syphilopathogenic nature of the spirocheta.

Schaudinn and Hoffmann[1] have discovered a Spirocheta in the various luetic lesions. They designated this spirillum on account of its refractivity to stain, *Spirocheta pallida.* The organism was discovered living and in smears stained by a modification of Giemsa's blood stain. Living it is a very fine, slightly refracting, actively mobile spirillum, thread-like and twisted in corkscrew-like spirals, pointed at the ends. Buschke and Fisher have found it in the internal organs of a child dead from congenital syphilis. It has been found by several syphilologists in blood from secondary and tertiary luetic dermic lesions. Mulzer[2] has found spirochetæ of various types at the surface of ulcerated carcinoma. Borrel has found similar spirochetæ in non-ulcerated carcinoma of the mouse.

Concerning these Hoffmann remarks that they not only resemble *Spirocheta refringens* but that often types occur difficult to distinguish from the S. pallida. The S. pallida has a length of from 4 to 14 *m.* and a breadth of 0.25 *m.* The S. refringens is larger and very easily recognized.

A. Fanoni,[3] of New York, has recently succeeded in *staining the S. pallida.* Scrapings were made from chancres with a sterile scalpel. The juice of inguinal glands and some tissue débris were obtained by puncturing with a large hypodermic needle, moving it about so as to break up the tissue. Condylomata were clipped off and squeezed to

(1) Berlin klin. Woch., May 29, 1905.
(2) Arch. gén. de Méd., July 25, 1905.
(3) Med. News, Oct. 6, 1905.

obtain their contents. The papules were incised through their centers and scraped with a Volkmann spoon. Smears were made in the usual way with this material, spreading them as thinly as possible. In staining them three methods were used. Giemsa's method as used by Schaudinn and Hoffmann is as follows: Fix in absolute alcohol for half an hour. Then immerse for twenty-four hours in the following solution: (1) Twelve parts of a solution of eosin (2.5 c.c. of 1 per cent eosin solution in 500 c.c. water); (2) three parts of azur I (1 part dissolved in 1,000 parts water); (3) three parts of azur II (0.8 parts in 1,000 parts water). Wash in water.

The method of Oppenheim and Sachs is as follows: The slides first dried in the air are placed in the following solution without fixation: 100 c.c. of 5 per cent carbolic solution in water; 10 c.c. of a concentrated alcoholic solution of gentian violet. Slide is dried slowly by gentle heating over a Bunsen flame until it begins to steam. It is then washed with water and dried with filter paper. The spirochetæ are *very distinctly blue and seemingly* larger than those stained by Giemsa's method, because the alcohol in the latter dehydrates and shrinks the germ. The process stains in a few minutes and is probably the best for clinical purposes.

De Marino's method consists in staining the slide without fixation with 1 c.c. of Marino's blue (0.10 c.c. of azur blue to 50 c.c. methyl alcohol). Allow this to remain for ten minutes, and without washing drop on the slide 1 c.c. of a watery solution of eosin 1:50. After two minutes wash and dry. The S. pallida will be stained a faint orange pink. On account of its rapidity this method is likewise preferable to Giemsa's, but the stain is not as distinct as that of Oppenheim and Sachs'.

According to C. Thesing,[1] the S. pallida *has no etiologic relation to syphilis.* The spirilla are in many instances not derived from the interior of the lesion but from the surface of the skin or from the stain, where many organisms occur unless it be boiled. Thesing found structures practically identical with the forms described by Schaudinn and Hoffmann in the smegma of absolutely healthy individuals.

(1) Munch. med. Woch., July 11, 1905.

A. Gordon[1] on testing the fluid obtained by lumbar puncture in eight cases of cerebrospinal syphilis failed to find the spirocheta. He expresses the opinion that perhaps in parasyphiloses, which are tertiary manifestations of lues, there is only a *chronic* intoxication produced by a toxic product elaborated by the spirochetas. Metchnikoff and Roux assert that positive results are obtainable only in primary lesions. McWeeney obtained positive results in primary lesions, but failed to find spirochetas in advanced tertiary ulcerations.

The morphology of the spirocheta clearly requires severely analytic study since, according to E. J. McWeeney, kala azar (a disease due to Leishmann-Donovan bodies, presumably a life stage of trypanosomes) is propagated by coitus. As Leonard Rogers[2] has shown, a trypanosome (T. equiperdum, Doflein) is the cause of *dourine,* or so-called "horse syphilis."[3] Though trypanosomes are conveyed usually by an invertebrate intermediate host, still they are conveyed from stallion to brood mare by direct contact. As Rouget has shown, they can pass through the unbroken mucosa. As a spirocheta has lately been found by Schaudinn to be a stage in the life history of a trypanosome of the stone owl (T. Ziemanni), McWeeney raises the question whether all spirochetes—for example, those of relapsing fever, pseudodiphtheritic angina and tropic spirillosis—are not likewise protozoa and mere stages in the life history of flagellates?

Siegel[4] claims the discovery of a *flagellate protozoon as the specific cause of syphilis.* This protozoon (the Cytoryctes luis) is an actively mobile flagellated body measuring from 0.5 to 2.4 *m.* in diameter. These bodies are fairly refractile, but can be seen only with the very highest apochromatic objectives enforced by strong compensating eye pieces and the best illumination. The larger forms are laterally somewhat flattened, pear-shape bodies, provided with a single flagellum at each end. In the smaller forms a flagellum occurs only at the tapering end. The organisms can be stained with a hematoxylin-azur mixture. The larger forms then show two distinct round nuclei, while in the

(1) Amer. Med., July 22, 1905.
(2) British Med. Jour., June 10, 1905.
(3) Practical Medicine Series, Vol. X, 1904.
(4) Med. News, July 1, 1905.

smaller bodies from four to sixteen can be seen. Although it is possible to demonstrate numerous parasites in various stages of development in almost all the tissues of thoroughly infected apes and rabbits, the most striking results are obtained about three weeks after primary inoculation with renal parenchymatous juice.

Jesionek and Kiolemenoglone[1] have *found protozoid bodies* in the kidneys, lungs and liver of a child dead from hereditary syphilis. These cells were 20 to 30 *m.* in diameter, and had round nuclei and granular protoplasm. Ribbert[2] has found *similar bodies* in the convoluted renal tubules of a child dead from syphilis and in the parotid gland acini of children whose death was not known to be due to syphilis.

Reinfection of syphilis is reported by H. G. Klotz[3] in the case of a German furrier who came under observation in 1882 with a large gummatous infiltration of the middle of the sternum and adjacent ribs on the right, several gummata of the right tibia and a number of strands and plaques imbedded in the external muscles of both sides. He admitted gonorrhea at 18, but denied chancre. The conditions present healed up under antisyphilitic treatment. Six years later the patient acquired a typical chancre, followed by secondary symptoms.

Successive syphilitic chancres have been studied by M. G. Sabouraud,[4] who finds that successive chancres often occur before the apparition of general symptoms. They result from inoculations either simultaneous or successive, which may be made before the appearance of the first chancre, or even after this. The infections made before the appearance of the first chancre are due either to the virus of the patient (auto-infection) or to the virus of another person (hetero-infection). If the successive chancres appear at more than ten days' intervals the question of auto-infection is suggested. The epoch of appearance of different chancres does not at all indicate the onset of infection or the date of incubation, for the incubation of successive chancres is variable not only in heterogeneous infection but likewise in autoinoculation. The latter does not depend on

(1) Amer. Jour. Med. Sciences, July, 1905.
(2) Zentralb. f. Allg. Path. u. Path. Anat., 1905.
(3) Boston Med. and Surg. Jour., Dec. 8, 1904.
(4) Arch. gen. de Med., Aug. 18, 1905.

the age of the chancre or upon the glandular state, but is subordinated to the appearance of general symptoms. Successive chancres appearing more than ten days before secondary symptoms are true erosive chancres. Those which show themselves in the last ten days previous to secondary chancres and their multiplicity neither advances nor aggrasymptoms remain papules or abort. The succession of vates secondary symptoms. The existence of successive chancres shows that immunity of the organism as a whole occurs but late in the secondary period. It seemingly occurs in part in the last ten days precedent to the secondary constitutional symptoms. This tardy immunity is due to the late diffusion of the virus in the blood. The virus is so late in entrance to the blood because it follows the lymph vessels and encounters glandular obstacles on its way. Diffusion by the lymphatics is proven by the following facts: (a) In case of head chancres, notably eyelid chancres, the glandular infection can be followed step by step toward the lymph collecting vessels of the base of the neck; (b) the onset of secondary symptoms is more precocious where the lymphatics have fewer glands to encounter or have a shorter course to the blood; (c) necropsies during the secondary incubation have shown the centripetal glandular route, which the lymphatics starting from the chancre follow in their course toward the great collecting canals (thoracic canal); (d) lymphocytosis which has been observed in the blood in the secondary period proves that the virus is then localized. On the contrary, upon the onset of secondary symptoms, when the virus is present in the blood, there is noted polynucleosis with alteration of the hematin; the syphilitic virus in following this centripetal glandular path immunizes in a measure and swells the glands of the regions it traverses. In other words, general immunity of the organism is preceded by a regional immunity. In spite of the tardy onset of immunity numerous chancre reinoculations have given negative results. These results are due to multiple causes of which the most important are: Regional immunity when the reinoculation has been made in the neighborhood of the chancre; general immunity when the reinoculation has been made too late; local treatment of the chancre, and, finally, mercurial treatment of the patient.

The pathologic process of *disseminated syphilis encephalitis,* according to A. M. Barrett,[1] is characterized by multiformity. In the brain there is a diffused degenerative process affecting the nerve elements, glia and blood vessels throughout the whole cortex. Apart from this there are disseminated areas of encephalitis, which are apparently not dependent upon changes in the meninges. They show early granulation tissue as well as local degenerative changes in the nerve elements, and secondary reactive changes in the glia. Hemorrhages, focal softenings and questionable gummata present are processes independent of these areas. The case Barrett thus describes was one of disseminated syphilitic encephalitis in a 41-year-old man, who was admitted to the insane hospital December 11, 1902, and died a week after. May 21 he was infected with syphilis and on June 12 had an initial lesion on the dorsum of glans. July 2 he was seen when recovering from a spree. The primary sore had taken on an ulcerative type and there was a papular eruption over the arms, chest, abdomen and legs. He had an attack of iritis, which disappeared under treatment. In November he had severe headaches, became neglectful of his personal appearance, forgetful and stupid and had to be fed. When admitted he was covered with an eruption of discrete scaly papules over the arms and maculopapular over the chest. In the mouth were mucous patches; the inguinal and epitrochlear glands were enlarged. He died a week after admission, having remained in a condition of stupor. Although the question is not raised by Barrett, it would be of interest to know how far alcohol predisposed to the disseminated condition found. Diffuse lesions occur in many types of chronic insanity; hence it would likewise be of interest to determine whether the conditions found were not those due to syphilis attacking an already diseased brain. Similar conditions have been found after the essential fevers and secondary to pertussis. This case destroys the validity of recent claims that syphilitic dementias are due to focal lesions of the frontal lobe alone.

Paretic dementia and syphilis have been co-related as to onset in 112 cases by A. Fournier.[2] The shortest interval

(1) Amer. Jour. Med. Sciences, March, 1905.
(2) Bull. Acad. de Med. de Paris, 1905, No. 7.

between the initial lesion and the onset of paretic dementia was three years. In four-fifths of the cases the psychosis developed between the sixth and the fifteenth year. In the majority it made its onset at the tenth. In thirty-seven cases reported by J. G. Kiernan[1] eight had the initial lesion five years before the onset of paretic dementia, eleven had it ten years and eighteen had it twenty years. In these cases the onset of the paretic dementia and the contraction of the initial lesion was exactly settled.

The cerebrospinal fluid is normal, according to Ravant,[2] in tertiary, dermic, osseous or mucous lesions. In eye lesions marked lymphocytosis occurs, greatest in recent cases but diminishing with lesion subsidence or atrophy. When an atrophic lesion is accompanied by lymphocytosis, nerve syphilis is still progressing. Headache, vertigo and mental disorder are sometimes accompanied with lymphocytosis. In old syphilitics, where the condition is quiescent, the cerebrospinal fluid is normal. Unexpected occurrence of cellular elements indicates examination of the nerves and eyes. In a syphilitic any cytologic reaction of the cerebrospinal fluid, isolated or accompanied with other luetic manifestations, indicates a neurosis if the reaction be traceable to syphilis alone. Lymphocytosis is an early indication of latent nerve syphilis, whose clinical evidence may not appear until long afterward. The cerebrospinal reaction in luetics is as characteristic a symptom as dermic or visceral lesions and has the same therapeutic indications. Lumbar puncture should be practiced systematically in luetics since its indications may lead to arrest of formidable lesions.

While in secondary syphilis intense and persistent dermic lesions are accompanied by lymphocytosis, the cerebrospinal fluid remains normal in tertiary dermatoses. Neurotic manifestations in secondary syphilis[3] are more frequent than Ravant believes and are often of brief duration, like the lymphocytosis. The onset of the secondary period is marked by more emphatic constitutional disturbances of the balance constituting health than is the onset of the tertiary.

Syphilitic diabetes, according to Troller,[4] may appear at

(1) Alienist and Neurologist, January, 1886.
(2) Ann. de Derm. et de Syph., December, 1904.
(3) Medicine, September, 1905.
(4) Arch. gén. de Méd., May 9, 1905.

the secondary period, but more often occurs at the tertiary. At the secondary period there often occurs a transitory glycosuria, which may in certain cases be transformed into a true diabetes. More often this glycosuria is referable to a nutritional disorder, while, according to Fournier, the true diabetes of the secondary period results from a cerebral localization. During the tertiary period there occurs three types: (1) A nervous diabetes appearing in individuals aged from 20 to 40 at a period comparatively approaching the onset of syphilitic infection (1, 2, 4 years) and accompanied with cerebral phenomena (fifth and sixth cranial nerve paralysis, facial paralysis, epileptiform crises, etc.). Sugar is never very abundant. The prognosis, though serious, is relatively favorable if treatment be promptly instituted. (2) A pancreatic diabetes appearing at a date very distant from the luetic infection (15 to 30 years). The onset is brusque and accompanied with gastrointestinal disorders. There is polyuria and a marked glycosuria. The stools are fetid and often contain fat. The prognosis is grave, death being the usual termination. There is sclerosis of the pancreas attacking secondarily Langerhans' islands. (3) Another diabetes, accompanied neither with cerebral nor pancreatic symptoms, appears from 15 to 20 years after the chancre in subjects aged from 35 to 50. The prognosis is more benign than either of the two preceding forms if treatment be promptly instituted. Its pathogeny is unknown. (4) A diabetes seemingly syphilitic by origin but not by nature; parasyphilitic diabetes is sometimes observed. Specific treatment is without effect, albeit the iodids exert a beneficial influence. (5) A heredo-syphilitic diabetes, curable under the influence of mercury, exists. It is due to the pancreatic lesions so frequent in heredosyphilis. In conjugal diabetes syphilis, according to Troller, is often found in the spouses. Syphilis may therefore often cause at once the conjugality and contagion of diabetes. In all forms described mercurial treatment should be rapidly instituted, which may lead to cure or arrest the evolution of the morbid phenomena, or may in certain cases act as a synergist to antidiabetic treatment, which should be prescribed at the same time. In the parasyphilitic type the iodids, as already pointed out, act better than mercurials.

Syphilitic metrorrhagia occurs, according to Ozenne,[1] generally during the tertiary period, but may appear during the secondary, and may be of diagnostic value in absence of local evidences of infection. Two of his cases yielded to mercurial treatment after local therapy failed.

Syphilis in the *second and third generation* is reported by C. Boeck.[2] Four children had coryza, maculo papular eruptions, scaling of the palm and soles, pigmentations and rhagades. The grandmothers had been treated for syphilis at Boeck's clinic. In one instance the mother had been treated for hereditary syphilis 29 years before the child's birth. The mothers had syphilis 20, 21, 26 and 29 years before the birth of the child. Paternal reinfection of the mother could be excluded. In Sweden hereditary lues from the father is rare.

Lucas[3] has had under observation *three generations* affected with syphilis. The grandmother, aged 55, had been treated for several years for gumma of the upper part of both legs. Her daughter at 16 was treated for congenital gumma of the popliteal space, and at 20 for pharynx ulceration, followed by nearly complete naso-pharyngeal stricture. A son which this girl bore at 22 had scalp ulceration soon after birth and nasal discharge which disappeared in a fortnight under antisyphilitic treatment. At 5 the child was normal but for prominent frontal eminences and depressed nose bridge.

Acquired syphilis in children was found by Glück[4] in 215 cases, or 6 per cent of the total number of cases of syphilis under 14. The primary lesion generally affected the lips, tongue, tonsils or other parts of the mouth. The genitalia were affected in three cases only. True chancre was present in but six cases. The primary lesion was usually a papular eruption. Lymphadenitis was a common symptom. Exanthemata were present in 32 per cent of the cases. Specific laryngeal affections were present in 50 per cent. Headaches, alopecia and plantar and palmar psoriasis rarely occurred.

Secondary syphilis at 6 years from coitus is reported by

(1) La Gynecologie, June, 1904.
(2) Berlin klin. Woch., September, 1904.
(3) British Med. Jour., March 11, 1905.
(4) Münch. med. Woch., 1904, No. 46.

G. F. Lydston.[1] The initial lesion had been contracted from a girl of 9 who had vulvar condylomata. Three months later the boy had generalized papulo-erythematous syphilides; some in the pigmentary stage. There was typical primary inguinal adenopathy. There was generalized adenopathy, the femoral and epitrochlear glands being especially prominent. Decided faucial engorgement was present; there were mucous patches on both tonsils. There was phimosis and a typical initial induration at the preputial orifice.

Colon lesions from congenital syphilis are reported by M. Alezais[2] which are characterized by a notable thickening of the submucous tissue. There is nothing found at this point but fibrous tissue with embryonic infiltration especially marked at the superfices and around the vessels. The lesions are related to those observed in hepatic syphilis without gumma.

Mitral stenosis, aortitis, coronarteritis and tabes *in a syphilitic woman* are reported by L. Renon.[3] The coronary arteries were completely obliterated. There had been frequent attacks of angina.

Sixteen cases of *syphilitic synovitis in children* are reported by Dunlop.[4] All were secondary to luetic epiphyseitis or primary synovitis in the knee joint. Most often the condition began with swelling of the synovial membrane. Later there were spongy granulations, and later still bone thickening. There was an insidious onset, chronic course, symmetric distribution, absence of pain and free passive mobility. There were luetic lesions elsewhere and there was decided response to antisyphilitic treatment.

Tabes, accompanied with florid manifestations of lues, has a better prognosis than tabes where such manifestations are absent. It is probable, however, that this form of tabes is merely one of the neurasthenic manisfestations of lues and not a true locomotor ataxia. In such case the luetic manifestations would be more florid than when the lues reached the parasyphilitic stage.[5]

The relation of the *offspring of tabetics to juvenile tabes*

(1) N. Y. and Phila. Med. Jour., Sept. 15, 1904.
(2) Arch. gén. de Méd., Sept. 12, 1905.
(3) Arch. gén. de Méd., April 4, 1905.
(4) Edinburgh Med. Jour., December, 1904.
(5) Zeit. f. klin. Med., LV.

is discussed by Milian.[1] The fecundity of tabetics is diminished. This diminution is due to syphilis rather than tabes. The small proportion of juvenile tabetics, the offspring of tabetic parents as compared with the offspring of syphilitic parents, indicates that hereditary syphilis is a direct cause of juvenile tabes.

Syphilitic hemorrhage occurred in 3,364 new-born children at the Philadelphia Lying-in Charity, according to W. R. Wilson,[2] forty-five times. The majority of the cases were fatal. Ten of these presented evidences of syphilis, either in the parents or in the children themselves. Hemorrhage appears in children who do not present characteristic signs of syphilis. When this rule does not always hold good congenital manifestations, such as pallor, rhagades, shrunken skin, are more often observed than the hereditary stigmata, such as the characteristic eruption and snuffles. The more distinct types of the latter form seem to make their appearance where infection is more or less recent, while in the hemorrhagic cases the bleeding seems to be the result of certain constitutional changes due to remote infection. Leakage may occur from the minute blood vessels in the skin, mucous membranes, serous membranes, meninges, brain, glands and organ parenchyma.

Syphilis of the placenta has been studied by C. Nelis,[3] who finds that syphilitic lesions of the placenta are nonspecific in character and are frequently observed in nonspecific cases. One feature, however, is rarely observed except in syphilis: villi hypertrophy, due to stroma sclerosis. It is obvious that, like all sclerosis, this might be produced by toxins of many microbes, and even by autotoxic products. It therefore affords, as Nelis remarks, a probability but not a certainty of the presence of syphilis. Nelis finds with Schwab in opposition to Frankel that placental lesions do not vary in position according as the lues is due to the mother or to the father. Luetic placental lesions are expressions of the struggle between mother and child. Villi hypertrophy shows that the child has required more air than it was getting, while the fibrous developments of the maternal parts show maternal attempts at blocking off the diseased part of the fetal placenta.

(1) Rev. des Malad. de l'Enf., March, 1905.
(2) Archives of Ped., January, 1905.
(3) L'Obstetrique, September, 1904.

Syphilitic nephritis is reported by Emanuel[1] in a 15-year-old girl who had Hutchinson's teeth, depressed nose and prominent frontal eminences and depressed chest. Eight months before coming under care she had facial eczema, albuminuria and increased micturition. There had been no infectious disease or traumatism. The ordinary treatment of nephritis was without effect. She recovered under potassium iodid despite unrestricted diet.

Luetic hectic is often *a symptom of visceral lues,* according to Klemperer.[2] It may result from hepatic, renal or pulmonary lues. It is of persistent type. John Hunter was of opinion more than a century ago that there was a monosymptomatic constitutional syphilis of which fever was the single symptom. Fever, according to A. Cecconi,[3] may accompany all the manifestations of tertiary syphilis and may be its sole expression. This possibility should always be taken into serious consideration when in presence of a fever whose cause cannot be determined. The luetic origin of fever cannot be excluded, according to Cecconi, except where specific treatment is without effect. This last conclusion is, however, too strongly put since syphilis may set in action metabolic changes disturbing the temperature regulating apparatus which would not yield to antisyphilitic treatment alone. These secondary non-specific results of syphilis are too much ignored, perhaps because the mercurials in addition to their specific effect exert an alterative action as in non-syphilitic subjects.

Luetic *incontinence of urine and feces* in hereditary cases have been discussed by Cantonnet,[4] who has analyzed twenty-one cases. Urine incontinence is sometimes due to meningomyelitis from late hereditary lues. There is at times fecal incontinence, particularly in degenerate and neurotic subjects. The condition occurs at all ages, but particularly between 4 and 20. There are two clinical forms; one with decreased or abolished reflexes and the other with contractures and exaggeration of reflexes. The prognosis is dubious because of later possibilities. The sphincter troubles are of favorable prognosis.

Buccal leucoplasia in relation to syphilis has been

(1) British Med. Jour., March 11, 1905.
(2) Zeit. f. klin. Med., LV.
(3) Rev. Crit. di Clin. Med., March 25, 1905.
(4) Rev. Francaise de Med. et de Chir., April 17, 1905.

studied by R. Trapenard,[1] who reports the view of Gaucher that buccal leucoplasia is always of syphilitic origin. Trapenard has collected 126 cases in which syphilis was determined to be present 116 times. In ten cases only the patients presented neither symptoms of syphilis nor syphilitic antecedents. In these cases hereditary syphilis or latent syphilis of exceptional origin could not be excluded. Certain leucoplasia occur in the course of secondary syphilis by transformation of mucous patches into leucoplasic patches. The greatest number develop at a late epoch, but even then often result from the transformation of late or relapsing mucous patches. Treatment should always be given, as it frequently improves the cases or at least arrests the progress of the disorder. Trapenard does not discuss at length the determining factors of leucoplasia to which Benard[2] called attention about two years ago.

In cardiac syphilis the myocardium is rarely affected alone or even principally, according to A. Foxwell.[3] The large arteries, the aorta and immediate branches, with the main divisions of the pulmonary and coronary arteries, are the chief victims. If with a large heart there is a healthy radial and no renal cirrhosis, but evidence of disease in the large arteries, then diagnosis of myocardial degeneration due to syphilis may logically be made.

Epidermic dryness and mobility as luetic stigmata have lately been described by W. P. Jukowski.[4] The entire skin is loose and covers the underlying cutis like a thin shirt. When the child moves this loose epidermis folds and presents a wavy surface. A few days after birth the dermic appearance changes owing to deep cracks and desquamation. The fissures involve the entire skin. The bleeding stripes and spots cause a variegated appearance. There is no icterus. In severe cases the children were aphonic, dying six days after birth. This would seem to be the result of arrested development, and what J. G. Kiernan[5] calls the simian or senile period of intrauterine life. This condition has been previously described by Souques[6] and E. S. Talbot.[7]

(1) Arch. gen. de Méd., Aug. 18, 1905.
(2) Practical Medicine Series, Vol. X, 1903.
(3) British Med. Jour., Oct. 21, 1905.
(4) Med. Obs., 1905, No. 7.
(5) Progrés Méd., July 15, 1888.
(6) Medicine, 1901.
(7) Degeneracy: Its Causes, Signs and Results.

Extra-Genital Chancres have been observed in 5 per cent of 1,217 cases of syphilis by D. W. Montgomery.[1] The percentage of other observers is as follows:

	Per cent.
Krefting (Christiania) reports	15.6
Fournier (Paris) reports	9.0
v. Broich (Germany) reports	9.0
Van Walsen (Amsterdam) reports	8.5
Mracek (Vienna) reports	7.5
Bulkley (New York) reports	5.5
Finger (Vienna) reports	1.3

In Russia from 75 to 80 per cent are acquired in this manner from popular customs. All statistics, except those of Neumann and Krefting, give an excess of males. Neumann gives 34 males and 48 females because of the great number of chancres of the lips and of the breast. Krefting gives 61 males and 231 females for the like reasons, infection by wet nurses and kissing. The disease he finds is also transmitted among the peasant class by eating out of the same dish. In 67 cases the following were the locations: Lips, 25; tongue, 3; gums, 1; tonsil, 1; corner of mouth, 4; cheek, 1; eye, 1; neck, 1; abdomen, 5; breast, 1; nates, 1; anus, 3; fingers, 7; wrist, 1; forearm, 2; back of hand, 1. Other observers found 56.4 per cent were on the lower lip; 43.6 per cent on the upper. In a case reported by Dudley Tait the chancre appeared on the stump of a Fallopian tube. A woman who had buccal syphilis dressed a sinus in this patient on whom Tait had operated for salpingitis, typical secondaries followed.

Palpebral chancre is reported by Leoz[2] in a 29-year-old woman who consulted him for an eyelid tumor which was round, hard and indurated. This proved to be a chancre. Chancre in this region is rare and may readily be mistaken for a chalazion, or even a simple blepharitis. Leoz has seen but two cases like this in 25,000 eye cases.

Chancre of the eyelid in a 21-year-old married woman has been reported by Mewborn.[3] About twelve weeks before coming under observation a swelling taken for a stye appeared on the left eyelid margin. A few days later

(1) Jour. of Cut. Dis., August, 1905.
(2) Siglo Medico, April 22, 1905.
(3) Jour. of Cut. Dis., April, 1905.

this was cauterized with copper sulphate and silver nitrate. Four weeks later the lid was enormously swollen and covered with a black crust. The ocular conjunctiva was edematous. The preauricular, cervical and submaxillary gland were enlarged. Four weeks later there was secondary roseola. At the time she came under Mewborn's care there was a dull crescentic papule at the center of the left lower limb, with some loss of tissue from sclerosis. The lashes were entirely absent. There were but two shot-like preauricular glands and enlarged cervical and submaxillary glands. The tonsils were very much enlarged and an elevated patch encroached on the right soft palate. The mode of infection in Mewborn's opinion was from the copper or silver nitrate stick employed in cauterizing the stye.

Mewborn suggests the disuse of copper or silver sticks in dispensary practice, and the employment of strong solutions on cotton swabs, which can be thrown away. According to Allen and Fordyce, eyelid chancre is far from infrequent.

Nose chancre has been reported by H. G. Anthony[1] in a young woman who had an ulcer on the left side of the nose, which was superficial, surrounded by a hard rim of indurated integument that elevated the border and caused the appearance of a deep ulcer. It had a regular outline, slightly oval form, and a diameter of .8 inch. It bled readily, and when squeezed blood and serum only exuded. Its base was red, smooth and shiny. The neck glands on both sides were enlarged in packets and painless. There was no eruption of the skin, buccal mucous membrane or genitals. Six weeks previously there was a papule in the location of the ulcer which the patient picked, after which the ulcer appeared and extended. Infection came from the girl's paramour.

M. L. Heidingsfeld[2] reports a case of *tonsil chancre* which developed in a patient four weeks after marriage, accompanied with marked enlargement of the cervical glands, producing deformity on the affected side. The ulcer produced extreme pain and distress and felt indurated. Five weeks later a second and post-initial chancre manifested itself on the right labium majus, which was

(1) Jour of Cut. Dis., April, 1905.
(2) Cincinnati Lancet-Clinic, Sept. 9, 1905.

accompanied by characteristic enlargement of the inguinal glands on the opposite side.

Sore throat and fever are frequently the first evidences of syphilis, especially in women, according to W. T. Belfield.[1] He reports two cases where recent syphilis of the throat in married women with husbands under care for syphilis was treated as diphtheria. In one antitoxin had been injected. In another case sore throat with fever was called "tonsillitis" until a pink eruption on the chest was noticed, whereupon the diagnosis was changed to scarlet fever and the house quarantined. A girl just recovering from alleged quinsy and canker sore—really syphilis of the throat and mouth—inoculated her lover's lip with a chancre. Examination confirmed her claim of virginity but revealed a scaly indurated papule on her left nipple. She had applied to this several times a sickly fretful child of a married sister which had mucous patches and snuffles; its father was syphilitic.

Tonsil chancre and throat disorder are frequently confounded in cases other than those cited by Belfield. Ohmann-Dumesnil[2] reports the case of a young girl in whom two weeks before coming under observation the satellite group of cervical glands had been removed as tuberculous. The febrile symptoms were of the hectic type described by Klemperer. In a male typhoid fever convalescent there was left on the subsidence of the typhoid symptoms tonsil chancre, general adenopathy and a maculopapular syphilide. Tonsil chancre and Vincent's angina were found coexistent in two females and one male. Diphtheria and active throat lues co-exist at times, according to Ohmann-Dumesnil.

Obstetric syphilis is reported by A. M. Crispin[3] in the case of a physician who suffered from a recurrent pompholyx which had ruptured while he was delivering a syphilitic woman. According to J. G. Kiernan[4], obstetrical ly acquired syphilis often lies behind paretic dementia in physicians. The finger chancre is not recognized and is allowed to affect the entire system of the obstetrician, who, being overworked, is peculiarly predisposed to syphilitic

(1) Med. Record, Dec. 10, 1904.
(2) St. Louis Med. and Surg. Jour., June, 1905.
(3) Amer. Jour. of Derm., January, 1905.
(4) Med. Standard, August, 1891.

nervous accidents. The first evidence of paretic dementia is a moral breakdown, which ruins the physician usually at the age of 50, long before its morbid mental source is recognized. *Finger chancres,* according to Fournier,[1] occur in 49 cases out of 10,000. Seventy-five per cent occur in physicians, and the prognosis is grave, as they long escape proper treatment. They are readily confounded with panaris. The symptomatic bubo may involve the epitrochlear glands or those of the axillary, or both at once. Finger chancres are, according to D. W. Montgomery[2], *peculiarly frequent in physicians.* The index is most frequently attacked. Next in order comes the middle finger, since it is allowed to rest on the lower lip while an instrument is being held in the mouth of a syphilitic patient or on the external genitalia when the index is inserted in the vagina. The thumb and little finger are next most exposed, while the ring finger is rarely attacked. Finger *chancre from intubation* is reported by Ohmann-Dumesnil[3] in the case of a physician inoculated by using the index finger in inserting the tube in a luetic child suffering from diphtheria.

Nipple chancre, as J. T. Priestly[4] has pointed out, is next in frequency among extra-genital chancres to chancre of the lips. Nipple chancre is far from infrequently due to titillation by men with buccal mucous patches.

Syphilis from shaving is reported by W. T. Belfield[5] in a 47-year-old man who came under care for a skin eruption. Typical recent syphilitic affection was found in the throat, tongue, skin and lymph glands. A small ulcer with indurated border and base existed below the angle of the left jaw. The genitals were free from disease. The ulcer had resulted from a cut by a barber, the bleeding from which had been checked with a styptic pencil. Two weeks later the cut, which had never healed, developed into the ulcer. There was marked cervical gland enlargement on that side. A 39-year-old man came under observation with an indurated ulcer on the chin, resulting from removal of an ingrowing hair, the bleeding having been checked with a styptic pencil. The glands on each side of the neck were

(1) Jour. de Med. de Paris, 1894.
(2) Jour. of Cut. Dis., April, 1905.
(3) St. Louis Med. and Surg. Jour., June, 1905.
(4) Medical Standard, 1894.
(5) Med. Record, Dec. 18, 1904.

much enlarged. Nine weeks after the inoculation, during which time there was continuous fever and a loss of 20 pounds in weight, constitutional syphilis became manifest in the throat, on the tongue and skin and in the lymph glands. The genitals were unaffected.

Zoniform syphilides, according to A. Collard,[1] are cutaneous lesions of the tertiary period clearly syphilitic by their morphologic character and evolution, but suggesting zona by their localization and disposition. They are formed of tubercular or papular elements, covered or simply bordered by squamous epidermis. As they belong to the tertiary period they are monomorphous, constituted in each case by a single eruptive modality. They attack the skin more deeply than secondary syphilides. They are not generalized but grouped in certain points of the integument. They display a tendency to unite in corymboids, in rings or in more or less rounded patches. They are apyretic, aphlegmasic, apruritic, and present either a ham color, or more rarily a copper. These syphilides have nothing in common with zona, but their localization on nervous territories and their disposition. Apyrexia and analgesia distinguish them from herpes zoster. While the last passes in ten or twelve days through the successive stages of erythema, vesicle and crusts, the zoniform syphilides appear from the onset as papules or tubercles, which become more numerous and confluent and recover very slowly, but more often become chronic. True zona in syphilitics presents its classical aspects. There are sometimes found in syphilitics eruptions which are neither true zona nor syphilides and which Jullien classes among parasyphilitic manifestations. These zoniform syphilides are produced like zona among syphilitics by the action of the pathogenic agent of syphilis or of its toxin on the spinal axis. According to the metameric hypothesis their localization shows that the segment of the cord which corresponds to the territory is affected by the morbific agent.

Circinate psoriasis around luetic cicatrices is reported by Hallopeau and Roy.[2] The case was not a hybrid one, but an instance where the cicatrices had served as a species of culture medium for the pathogenic agent of the psoriasis.

(1) La Pressé Méd., March 25, 1905.
(2) Arch. Gen. de Med., Aug. 1, 1905.

The condition was similar to the part played in psoriasic localization in the same patient by vaccination cicatrices and similar traumatisms.

Luetic infection of paretic dements is discussed by J. G. Kiernan,[1] who points out that the experiments of Krafft-Ebing on inoculation of paretic dements with syphilis have two great elements of error. Syphilization, as Auzias-Turenne has shown, does not always succeed even though the person inoculated has never had syphilis. On the other hand, syphilitic reinfection does occur. This would not unlikely be the case in the luetic paretic dement where syphilis has lost all its specific characteristics and has become simply a parasyphilosis. In most of Kiernan's cases syphilis prior to the paretic dementia could be positively excluded. In the others there is no positive evidence of its existence. In four of the cases reported the initial lesion was extra-genital. Ripping and Snell[2] demonstrated twenty-five years ago that syphilis was sometimes merely an epiphenomenon of paretic dementia concomitant on the sexual excitement of the earlier stages of the psychosis.

Luetic keratoderma simulating erythema keratodes is repirted by A. Garceau.[3] A 33-year-old man had, since he was 8 years old, at intervals of from four to five years, recurrent erythema of hands and feet, followed by accumulation of scales. All the attacks prior to the one for which he consulted Garceau had disappeared under local treatment. The last attack had incapacitated him from work for 8 weeks prior to this consultation. Redness and nodules first appeared about the finger joints, which spread in patches and finally coalesced in a large diffused patch on the palms. The erythematous area on the left hand extended over the entire palmar surface from an inch to an inch and a half of the wrist well up and covering the entire palm, thence up to the first phalanx. The thick incrustation was nodulated at the phalangeal, the metacarpo-phalangeal articulation and at the wrist. The area on the right side was not so extensive. Islands of normal tissue traversed the erythematous patches upon the dorsum. The incrustation extended between the index and middle fingers on both

(1) Medicine, October, 1905.
(2) Allgem. Zeitschr. f. Psych., XXXVIII.
(3) California State Jour. of Med., September, 1905.

hands and upon the middle phalangeal pairs of the right ring and middle fingers and the metacarpo-phalangeal articulation of the left little finger. The feet were attacked at the same time. There was a history of a soft chancre three years previously, but without secondary symptoms or antisyphilitic treatment. There was cicatricial tissue of a hard preputial sore. There was a congenital hypertrophied tongue. The hands and feet were edematous and covered with a thick horny growth. The nails were markedly pale with tenderness and pain on pressure. Microscopic examination of the horny erythematous tissue showed no difference from ordinary angio-keratomata. The case yielded to intramuscular injections of a 1 per cent solution of mercury sozoidolate for four consecutive weeks.

He reports another case involving both hands and one foot. The palms of both hands and the palmar surfaces of the fingers were hyperemic, and showed an overgrowth of horny tissue. On the arch of the right foot extending to the inner malleolus was a large erythematous spot covered with horny tissue. These conditions yielded to Lassar's paste locally and 15 grains of potassium iodid internally, *t. i. d.*

In syphilitic spinal paralysis, according to W. J. Dougherty,[1] the time that has elapsed between the chancre and the onset of symptoms indicating spinal cord involvement must be taken into consideration. Those cases which develop within a few years after the luetic infection must be looked upon—though slow in onset—as a secondary or tertiary expression, while if a considerable time has elapsed before the onset of cord symptoms, the condition must be referable to what Dougherty calls the "terminal syphilitic stage." The latter is evidently a parasyphilitic sclerosis such as was pointed out by Luys[2] more than a quarter of a century ago. Dougherty, in opposition to Erb, takes the very sound position that the distribution and dissemination of the lesion determines the paralysis rather than the etiologic factor.

Peripheral Nerve Syphilis. J. Grinker[3] reports the case of a man, aged 40, with history and traces of anterior syph-

(1) Med. Record, Aug. 5, 1905.
(2) Maladies Mentales, 1801.
(3) Medical Recorder, September, 1905.

ilis, suffering with slight trigeminal neuralgia, right-sided peripheral facial paralysis and neuritis of left sciatic and left anterior crural nerves. The combination of nerve affections pointed to syphilis as their cause, though two months' vigorous antisyphilitic treatment failed.

Tonsil staphylococcic infection mimicking chancre is reported by D. W. Montgomery.[1] There was a seemingly crateriform ulcer on the left tonsil. Its floor was yellowish, and its edges rounded and raised. The left anterior faucial pillar bulged forward, rounded smooth, and of a deep red color. The ulcer had extended slightly downwards. The sore on palpation was fairly firm, but not markedly hard. The ulcer was not painful, but caused much expectoration, which irritated it. There was a smooth, firm, but not hard, lymph gland behind the left ramus of the jaw. The left epitrochlear gland was enlarged but soft. The only organisms found were staphylococci. There was no fever and the general health was good. Under the local use of hydrogen peroxid and 30-drop doses of tincture of iron chlorid the ulcer and the gland enlargement disappeared in a week. Four weeks later a similar attack occurred. The resemblance to a chancre was then explained by the peculiar shape of the affected tonsil crypt, which was deep, funnel-shaped and had bulged out rounded rim which contained a dirty gray mass simulating the necrotic floor of a chancre, while its raised rim resembled induration. According to Mendel, this ulcerating chancriform amygdalitis often mimics chancre. Fournier designates it "lacunar or cavernous amygdalitis." The deep tonsillar crypt became filled with a cheesy mass which was infected by staphylococci, just as the sebaceous plug in a sebaceous gland often is in acne.

Luetic infection by toothbrushes is reported by A. Fournier.[2] A husband contracted a chancre. Mild constitutional lues followed. His very anemic wife was infected, who had severe secondary syphilis. The wife's sister and her babe came to live with the couple. She placed her tooth brush with her sister's, and later had her teeth extracted. She used water only with her tooth brush. She was infected and infected her child. Similar infections have been

(1) Canadian Practitioner, October, 1905.
(2) Bull. Gen. de Therap., April, 1905.

prevented by alcoholic or antiseptic dentifrices, according to Fournier.

Mammary syphilis mimicking breast cancer is reported by E. Beer.[1] The patient was a 35-year-old married woman, syphilized by her first husband some years before she became pregnant, and a rash occurred on the arms. "Lumps" occurred on her head and ulcers on the vulva. The child was prematurely born. She had been troubled ever since with sore throat, pains in the back, knees and shins. A short time before she came under Beer's care a mass had appeared in her left axilla and some weeks later a similar mass in the upper half of the left breast. Cancer was diagnosed by six physicians and operation advised. When she came under Dr. Beer's care she was anemic, nervous and thin, but not cachectic. In the upper half of her left breast was a movable non-tender unattached mass measuring 2 by 3 inches. The borders were indistinct, the surface smooth, and the mass was moderately soft but fluctuating. The nipple was normal. Directly below the middle of the left clavicle, apparently lying under the pectoralis major and pushing it forward, was a similar mass about half the size of the breast tumor. The borders were more distinct and it was rather spherical. There was another soft mass the size of a hen's egg in the left axilla freely movable and not tender; the same axilla contained a number of enlarged glands of the usual inflammatory consistency. The left supraclavicular region contained another mass of the same size and several small seemingly chronically inflamed glands. The right axilla and right supraclavicular region contained enlarged lymph-nodes of normal consistency. The posterior chains of cervical lymph-nodes of both sides were enlarged. While the general appearance was that of cancer, the condition of the glands suggested syphilis. The patient was put upon mercury hypodermically and iodid internally in increasing very large doses. Under this treatment the tumors at the end of two months and most of the glandular enlargement disappeared.

Intramuscular *injections of insoluble mercury preparations* in syphilis are, according to H. G. Klotz,[2] the best

(1) Med. News, Oct. 28, 1905.
(2) Ibid., April 1, 1905.

method of treatment. He has given some 2,500 injections and has had no bad results. He has given in various cases from 1 to 42 injections, usually suspended in oil or liquid vaseline. His custom is to give four to six series of six or eight injections at a week's interval and then his patient has all that is necessary. In some cases not more than 10 grains have been employed in all. In most cases the limit has been about 40 grains. Of late years he has discarded the use of iodid and has treated tertiary manifestations by mercury alone. He has often seen relapses that took place under mixed treatment become better under the injection treatment almost at once and then remain thoroughly under control. He agreed with the specialist in syphilis who said that one injection of calomel is sufficient to differentiate malignant disease from syphilis. Calomel injections are given deep into the buttocks. Nodules rarely result. There have been four abscesses in 2,500 cases. Where there is much going up and down stairs, the injection treatment is contraindicated. In cases complicated by tuberculosis the results are not favorable. Albuminuria is not produced by the injections nor aggravated by them. Diabetes is favorably affected.

In the discussion of Klotz's views, E. L. Keyes remarked that he had found intramuscular injections of mercury salicylate of great service. Syphilis should not be made too light for the patient, as otherwise the general health is neglected, which encourages the development of quarternary symptoms. In fulminant symptoms mercurial injections are of value. P. A. Morrow found in obstinate syphilis the injection treatment of great value. It should be a reserve method, not routine treatment. Where potassium iodid idiosyncrasy exists, mercurial injections are indicated. Mercury salicylate is the best form in which to give injections, though in some late lesions of the nose and throat calomel is most effective. Fuller was of opinion that the salicylates may be irritant, but were efficacious. The injection method is indispensable for quick results.

Mercurial treatment is of *decided value in the sphincter troubles* of late hereditary lues, according to Cantonnet,[1] who states that mercury is well absorbed in all forms,

(1) Rév. Française de Méd. et de Chir., April 17, 1905.

whether by inunction, by the mouth or by injection. Injections or frictions are preferable. Potassium iodid is useless. Stomatitis should be watched for, the child should be aroused at night to urinate and should not be permitted to overeat, but mercury is all-important.

Mercury lactate in syphilis has had good results, according to A. Bouveyron.[1] In the earlier stages and later when the oral mucous membranes are effected he prescribes pastilles of 0.075 grain of mercury lactate which are to be dissolved in the mouth after eating. Six of these are given per diem. They have very little metallic taste. The effect in bucco-lingual syphilides is very decidedly well marked. The mucous patches become less painful and heal quickly. The throat syphilides are less favorably affected, though the dysphagia becomes less distressing. The pastilles act well in mercurial stomatitis because of their bactericidal effect.

Untoward effects of potassium iodid are, according to A. Lieven,[2] avoided by the following modification of Spencer's formula:

℞ Pot. iodid 3viii
Ferri et ammon. cit.....3i
Strychn. nit. .....gr. 1-3
Elæosacchar. menth. pip.....gr. 85
Aquæ aurant. flor. q. s. ad.....3xxxii

M. S. 3i in a pint or more of water.

Potassium iodid, according to H. D. Singer,[3] should never be given in syphilitic disease of the vessels of the cord or brain, since being given with the hope of inducing blood coagulation in aneurism, it is obviously contraindicated where the object is to prevent thrombosis. According to Gowers, cases occur in which the iodid has precipitated the thrombosis and consequent hemiplegia which it was given to prevent.

Serum therapy in syphilis has been proposed by F. J. Bosc[4] through an analogy drawn between sheep pox and syphilis. The serum is prepared from the blood of sheep previously injected with luetic blood. The harmlessness of the serum determined, Bosc injected it into the patients before and after the appearance of the secondary eruption.

(1) La Semaine Med., 1905, No. 3.
(2) Med. Bull., October, 1905.
(3) Med. Herald, September, 1904.
(4) La Semaine Med., 1905, No. 2.

In the first class, while the rash was not, as a rule, prevented from appearing, it consisted merely of macules and was delayed beyond the usual time. One patient injected 18 days after the initial lesion had no eruption two months later. The dose of the serum is 5 grams.

Leredde[1] advises, in intractable syphilis, the use of *mercury benzoate or biniodid* in doses of a grain hypodermically. The dose carefully increased does not cause stomatitis.

Adjunct medication in syphilis has a considerable place, according to H. B. Hollen.[2] The general health of the patient and concurrent defects must be taken into consideration. Dietetics and digestives must be employed where the alimentary tract is disordered. Tonics are required where anemia and debility are present. Iron may be given in the form of the arsenate, carbonate or peptonate. An easily assimilable chalybeate are the nucleoids. Strychnin, gentian, hypophosphites, glycero-phosphates, cod-liver oil, arsenic, manganese and quinin all have important places in syphilotherapy.

Zittmann's decoction, according to G. H. Sylvester and C. B. Crisp,[3] has much more effect in constitutional luetic skin and connective tissue lesions than in luetic arthralgia.

Preventive Treatment of Syphilis. The weight of German army medical opinion is against this, according to C. E. Pollock,[4] because of the difficulty of diagnosing lues from the appearance of the chancre alone. Early use of mercury does not prevent ultimate development of the disease, or even shorten the treatment necessary to cure. Secondary symptoms yield more readily to mercury when its administration is delayed.

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(1) Rev. prat des Malad. Cut., 1904, No. 6.
(2) Medicine, February, 1905.
(3) Jour. Roy. Army Med. Corps, April, 1905.
(4) British Medical Journal, Oct. 14, 1905.

CHAPTER VI.

ACTINOTHERAPY AND RADIOTHERAPY.

The untoward effects of radium still continue to attract most attention. Piffard, in a discussion of radium effects, claims that while at times it arrests development, it also (as shown in the production of parthenogenesis) stimulates it. This, remarks *Medicine* (1904, p. 89), is an instance of the very frequent error which ignores the fact that balance, not excess in one direction, constitutes the normal. It ignores also the struggle for existence between the organs demonstrated by Roux.[1] Resumption of a function normal in animals, but ordinarily absent in man, is an expression of degeneracy, not of advance, since it destroys the physiologic balance previously existed. Parthenogenesis in man or mammals is a degeneracy, being an abnormal stimulation of a disappearing structure of the ovum. Reproduction has evolved from unicellular through complex parthenogenesis to bisexual types. During the passage of the ovum from the parthenogenetic type to the bisexual, degeneracies occur before the bisexual ovum becomes fitted for impregnation. A structure of the ovum (which Boveri has called the centrosome) atrophies, disappears and is replaced in function by a subtle osmosis between the sexes in certain infusoria, or at a higher stage by the spermatozoon. In certain invertebrates (as Loeb has shown) normal salt solutions will supply nutriment sufficient to replace the function of the centrosome. It is obvious on ordinary principles governing the stimulation of physiologic atrophies that radium simply stimulates the centrosome at the expense of higher development and hence constitutes an arrest of type development in the ovum. What is true of the untoward effects of radium is likewise true of the x-ray.

X-ray *sterility,* according to F. T. Brown,[2] occurs from exposure to an x-ray atmosphere. Azoospermia was found in 10 individuals who had been working with the x-ray

(1) Die Kampf der Theile im Organismus.
(2) Arch. of Roentgen Ray, March, 1905.

about three years, and in whom neither venereal disease nor traumatism accounted for the change. In a case of a patient treated for pruritus ani by the x-ray, his previously active spermatozoa disappeared and for three months could not be found. At the end of three months the normal condition gradually returned.

X-ray treatment of hypertrophied prostate has had remarkable results, according to Moszkowicz,[1] when the x-ray is applied through the rectum. In all cases three sittings of from 10 to 18 minutes each markedly improved the enlarged organs and the symptoms. The prostate subsided in size and urination markedly improved. In three cases the patients had evidence of arterio-sclerosis and angina pectoris.

Chronic x-ray *dermatitis,* according to J. Hall-Edwards,[2] occurs on the hands of x-ray workers. It is practically an occupation disease and creates a predisposition to the trouble. A few weeks' exposure to a weak radiation does more harm than a month's rest does good. There is some indication of secondary neuritis.

X-rays and the normal epidermis and epithelioma have been studied by Dalous and Lasserre,[3] who find that special lesions follow the x-ray which may be designated "radio-epithelitis." Similar modifications result in neoplasms. The epithelial cells are not equally affected by the x-rays; some are much more sensitive to the action of the rays than others. The cells in healthy epithelium composing the generative layer, and those immediately above them, and the corresponding cells in epithelioma are much more sensitive than the prickle cells and the cells of the corneous layer. The spino-cellular epitheliomata are less favorably affected than the vaso-cellular. The elective changes produced by x-rays in certain anatomic elements of the normal epithelium explain these facts.

The x-ray in hyperidrosis axillae has had decided effects, according to G. H. Stover,[4] who reports a severe case in a young married woman. The fluid poured out, saturated the clothing to the waist and dripped, staining clothing. The quantity was 2 quarts daily. The condition had ex-

(1) München. med. Woch. B., LII, No. 29.
(2) British Med. Jour., Oct. 15, 1904.
(3) Arch. gen. de Med., July 25, 1905.
(4) Arch. of Physiologic Therapy, April, 1905.

isted for years despite all treatment. A low tube 4 inches from the skin, exposing each axilla on alternate days, for ten minutes, was employed. After the third exposure the skin was slightly pigmented. After the twenty-fourth exposure there was slight erythema; at the thirty-first exposure perspiration was less, and at the thirty-eighth the improvement was marked. At the forty-fifth the hair fell out rapidly, but never became gray or white. The treatment was then intermitted; the perspiration being rather in excess. Four months later the axillary hair had returned. Twelve exposures were then given on succeeding days. After the eighth pigmentation occurred and the hair fell. At the end of the series slight erythema was present and the perspiration had entirely stopped.

Radium treatment of lupus, angioma, nevi and keloid scars has had good results, according to Werner and Hirschel.[1] They rayed with radium daily or every other day for several hours over a period of from three to fourteen days. Resultant scars were white, smooth and satisfactory. Recurrences had not appeared in from six to nine months after.

The high frequency spark in a xanthomoid degeneration of the lips has been found of value by C. W. Allen.[2] The condition first described by J. A. Fordyce[3] consists of a yellowish white discoloration of the vermilion border, chiefly confined to the epithelial cells; the protoplasm having degenerated into a substance allied to—but not identical with—keratohyalin. The condition is quite common, and, in Allen's opinion, is related in many instances to lues, although it may occur without this. In applying the high frequency spark he employs a static machine and a hyperstatic transformer, a large carbon electro, a spark gap of 2 inches, and a contact spark of about an eighth of an inch, or one which will cause almost at once marked whitening of the surrounding pink of the lip. Two or three sittings usually suffice and the pain is trifling.

The cold iron light (triplet or dermo lamp) in alopecia areata has been found of considerable value by Kromayer.[4]

(1) Münch. med. Woch., July 25, 1905.
(2) Med. Record, Sept. 23, 1905.
(3) Jour. of Cut. Dis., November, 1896.
(4) Deutsche med. Woch., 1904, No. 31.

The rationale of the treatment is artificial inflammation; the skin is irritated by frequent use of the lamp until marked inflammation results, when rest is ordered and the same treatment begun again. He cites six cases rebellious to other treatment which yielded to the lamp.

Jay F. Schamberg[1] reports the successful treatment of an intensive case of Lupus Vulgaris with the x-ray. Patient, a 31-year-old woman, with a tubercular family history, first showed lesions on inflated side of face, Feb., 1898. In 1891 the patient weighed 140 pounds; in 1899 she weighed 98 pounds. In 1903 a gland became enlarged and later underwent suppuration with necrosis of the overlying skin. No physical signs of tuberculosis of the lungs have ever been present. Plate 4 shows condition of patient at the beginning of the x-ray treatment March 31, 1903. In all, three hundred exposures were given in the course of almost two years. Plate 5 shows the condition of the patient in April, 1905. The cosmetic result is excellent.

(1) Journal Cutaneous Diseases, September, 1905.

Plate IV

CHAPTER VII.

GENITO-URINARY MEDICINE AND SURGERY.

Patent urachus is described by Swirt[1] in the following cases: A 58-year-old woman, who entered the hospital because of urine retention while urine dribbled from the navel. Artificial vesico-vaginal fistula was established. The urachus spontaneously closed, as later did the fistula. A 17-year-old girl dribbled urine constantly from the umbilicus. She menstruated from the same opening. The fistula edges were split and purse-string sutured, which resulted in union by first intention. A 1½-year-old boy had umbilical eczema with offensive urination from the navel. There was marked phimosis. Circumcision was without result. There was deficiency of the recti muscles. The umbilicus was excised; the urachus was closed with a purse-string suture.

Vesical Complications of Appendicitis.[2] If the appendix be in direct contact with the bladder, the inflammation becomes directly continuous with its wall; the same occurs if the abscess produced through the appendical inflammation comes in contact with the bladder wall. If the diseased appendix be any ways close to the bladder, the prevesical connective tissue becomes affected and a pericystitis arises. If the process continue, the bladder wall becomes affected and small hemorrhages of the vesical mucosa arise. If an abscess exist, it may rupture into the bladder; before doing so ulcers and ulcerating polypoid growths of the vesical mucosa form. After the rupture, fistulæ remain, most commonly on the posterior surface. Pus and feces may be excreted with the urine, and stone formation has occurred. Spontaneous closure of the fistulæ has been reported. G. Baradulin reports three cases of vesical complications during appendicitis. In the first the patient had four distinct attacks of appendicitis; during the second

(1) Zentralb. f. Gyn., 1904, No. 41.
(2) Amer. Med., July 22, 1905.

attack the abscess ruptured into the bladder, the fistula closing spontaneously; with the fourth, after a period of vesical pain and cystitis, the urine contained much pus for a time and also had a fecal odor. The patient suffered reflex vesical pain before the vesical wall was affected. The urine suffered no change of any kind until the abscess had ruptured and the rupture manifested itself through violent pain and sudden discharge of pus. In the second and third patients the symptoms never went beyond pericystitis, as proved by the frequent micturition associated with a normal state of urine. In both cases the appendix was found adherent to the prevesical structures; recovery occurred after operation.

Female *pseudo-hermaphrodism* is reported by G. E. Brewer[1] in the case of a 17-year-old patient who sought relief from a supposed left-sided inguinal hernia and undescended testicle. The patient was dressed in male attire and was by trade a barber. The face was of feminine type, the breasts were well developed and the figure that of a woman rather than a man. There was a rather undeveloped penis with no scrotum or testicles. The external urethral orifice was just below the penis in a cleft resembling either a bifid rudimentary scrotum or partially developed labia majora. The prepuce was redundant above and separated below, suggesting hypertrophied labia minora. There was no sign of a vagina. In the left inguinal canal was an oval swelling which could be pushed into the abdomen, but returned when pressure was removed and had a well marked impulse on coughing. Above the pubes to the left of the median line was an oval abdominal tumor about the size of a cocoanut; smooth on the surface and freely movable. The patient, when about 13 years of age, noticed occasional attacks of pain and discomfort in the lower abdomen at irregular intervals, which disappeared in a few days without treatment. She was not aware of the abdominal tumor. Brewer was of an opinion that the individual was a female with a hypertrophied clitoris and absence of the vagina. The abdominal tumor was an hematometra and the inguinal tumor an ovary. The mass in the inguinal canal was found to be

(1) Amer. Jour. of Urology, March, 1905.

a rudimentary uterus which contained a small amount of thick fluid blood. The large tumor was an enormously distended left Fallopian tube which contained considerable thick fluid blood. It showed on its surface, on microscopic examination, a considerable amount of ovarian tissue. The left broad ligament was attached to the peritoneum covering the sacrum. There was no trace of a right broad ligament tube or ovary. As the individual had always passed as a male, and there was no hope of bringing about conditions which would render a change in sexual status desirable, the uterus and tube were removed; uninterrupted recovery in three weeks.

Cryptorchidism has been found by W. B. Odiorne and C. C. Simmons[1] in 77 cases. Orchidopexy was performed 7 times in children aged from 5 to 13; in but two cases was a good result obtained. This was in a patient aged 13, who had double retention. Both testicles are normal in size, position and probable function. In two other boys operated upon at 13, both testicles have remained in the scrotum, but have retracted high up, although there is no notable atrophy. In one patient the testicle atrophied markedly, and remained fixed just outside the ring, where it caused constant pain. In two 5-year-old cases the results were doubtful. In one, the testicle withdrew into the canal, where it failed to develop. In the other it retracted into the pubic region. The operation was performed 11 times in cases aged from 16 to 42. Five testicles have remained in the scrotum. Four occupy a high position. One is in good position and half normal in size, but the seat of inflammation from traumatism. Three testicles retracted soon after operation into the canal, where they atrophied, one becoming painful. Two have retracted to the pubic region, where they cause much annoyance. In every case where the organ appeared normal at the operation it subsequently diminished in size. In two cases symptoms were removed; three were unaffected and two were aggravated.

Castration in sexual inversion, as Féré[2] points out, has been proposed to relieve social inconveniences, to prevent heredity and to cure the inversion. The fact, however,

(1) Annals of Surgery, December, 1904.
(2) Rev. de Chir., March, 1905.

that eunuchism is often attended by sexual perversion should have a tendency to limit the operation. Féré reports a case where castration was performed, and the testicles were replaced by two foreign bodies of the same size and consistency. The patient believed that his aspect and expression, sexually speaking, had been altered. When the wound was healed and the dressing removed, palpation of the prostatic apparatus shocked him. Frequent palpation occurred, leading to irritation, so that the removal of the foreign bodies became necessary. The patient was distressed at the emptiness of the scrotum, at the loss of voice which he thought indispensable to virility and intelligence and at the notion that everybody stared at him. He was subject to the same temptations as before the operation and had less power of resistance. A tendency to suicide present before the operation left him, but opium habit supervened.

Inversion is generally the expression of arrest of constitutional development of sex psychic and physical aspects, as Kiernan has pointed out.[1] The case of Guy Olmstead,[2] which came under judicial adjudication, illustrates castration dangers in inversion especially where there is congenital defect and general nerve instability. The man at one time had been in an insane hospital for querulent paranoia. Six years later, becoming a letter carrier, he developed a passion for a fellow letter carrier, to whom he wrote amorous letters, for which he was discharged. Two years later W. T. Belfield removed his testicles, which was followed by an irritable suspicional state. Erections, masturbation and the inversion continued as before. Later he shot the letter carrier to whom he was attached, but his prompt seizure prevented suicide. The recovery of his victim led to his incarceration for a short time in the Chester criminal insane asylum. When discharged from this he returned to Chicago and demanded his testicles from the city postmaster, whom he accused of being in a systematized conspiracy against him, dating from before the castration. The enormous mental element in inversion indicates its cortical seat, and should be prohibitive of any attempt to remove it on the reflex or local sexual theory.

(1) Alienist and Neurologist, 1901.
(2) Havelock Ellis: Sexual Inversion.

Enlargements of the testis and epididymis occur, according to D. N. Eisendrath,[1] in the following types: (a) Acute: (1) Gonorrhea almost invariably involves the epididymis, seldom the testis body or the testicle proper. (2) Trauma almost always involve the body of the testis proper and but seldom the epididymis. (3) Epidemic parotitis or mumps generally affects the testicle or the testis body proper. (4) Cystitis of non-gonorrheal origin is a frequent secondary complication, as also is inflammatory enlargement of the epididymus. (b) Chronic: (1) Tuberculosis is the most frequent cause of chronic enlargement, which usually first involves the epididymis, especially its tail, and later the body of the testis proper. It causes a marked nodular firm condition of the epididymis, often extending up the vas deferens. (2) Syphilis generally involves the body of the testis proper and rarely the epididymis. Sometimes tertiary syphilis involves both. (3) Tumors of the testis are benign or malignant. The first are usually adenomas or chondromas, while the malignant are usually sarcomas.

The testicle interstitial cells are so called because they are situated in the tissue intervening between the seminiferous tubules. According to A. Branca,[2] they are connective tissue cells so differentiated as to be capable of glandular action. The principal products of such action are fat, crystalloids and pigment, which last is more abundantly produced during the decline of life, when the reproductive power has become enfeebled or has been lost. The other two products, however, occur in profusion only during genital activity. Branca seems inclined to credit the cells with the action Brown-Sequard attributed to the testicle as a whole, but the testicle[3] must have a masculizing influence on the system long before their reproductive function is established, prior to the period of full development of the interstitial cells. The cells are present in the embryo, but so closely connected is their functional activity with the genital efficiency of the testicles that they undergo temporary atrophy during an animal's hibernation. In spite of their partial abeyance in early and late life, their pres-

(1) Chicago Med. Recorder, September, 1905.
(2) Presse Medicale, Aug. 12, 1905.
(3) N. Y. and Phila. Med. Jour., Sept. 23, 1905.

ence at a stage of embryonic existence preceding the differentiation of the sexual glands into testicles and ovaries has given rise to the hypothesis that they are the cause of that differentiation.

Untoward effects of urethral use of adrenalin are reported by W. S. Johnson.[1] A patient whom he had been treating for stricture and in whom sounds had been passed for several months came in his usual good spirits and in perfect health. After passing a No. 27 F. sound there was some bleeding from the urethra, which Dr. Johnson tried to check with a solution of adrenalin chlorid 1:4000 injected into the urethra with an ordinary glass syringe, filling the entire anterior urethra. The following result occurred almost instantaneously: Lividity, eyes set and glassy, patient crying out, complaining of bursting sensations in his abdomen, chest, neck and head, saying the top of his head would burst open, tingling and pressure in hands, arms, legs and feet; projectile vomiting, followed by complete collapse without loss of consciousness, lasting for ten minutes, respiration shallow, pulse imperceptible and heart sounds inaudible. Restoratives (strychnin, whisky and aromatic spirits of ammonia) were used hypodermically. While the patient gradually recovered, the dizziness continued to bedtime.

The meatus in *urethral stricture,* according to F. S. Watson,[2] must be cut to a caliber 1 mm. larger than that of the normal urethra previous to properly carrying out any treatment. Internal urethrotomy yields the most permanent results of any method, and for strictures of the anterior urethra, irrespective of their character, is the operation of choice. All resilient, intractable, nondilatable and impassable strictures of the bulbo-membranous or membranous urethra are best treated by external perineal urethrotomy. With the exception of these, all strictures so situated are best treated by gradual dilatation, unless, during its course, constitutional disturbances of importance arise, in which case it is safer to divide them at once by an external perineal urethrotomy. Divulsion and electrolysis are not to be recommended.

Watson points out that the *urethral calibre* is not natur-

(1) Jour. Amer. Med. Assoc., Oct. 7, 1905.
(2) Boston Med. and Surg. Jour., Dec. 8, 1904.

ally the same, nor can the meatus be taken as a guide to the urethral caliber. The average caliber is 32 (F.), but there is much normal variation; more above this figure than below it. The average meatus measures 22. There is a fairly constant relation between the circumference of the flaccid penis and the caliber of its urethra. Each ¼ inch in the increase of the circumference means a corresponding increase of 2 mm. in caliber. In treatment, Watson employs webbing bougies sterilized in a saturated solution of rock salt up to 18-20 F. and from then on, metal sounds.

Urethral papillomata, according to G. W. Allen,[1] are generally much softer than the ordinary preputial warts, although sometimes comparatively tough. The symptoms are those of gleet. He has observed a case of papillomatous urethritis in a 30-year-old man, who, 8 years before coming under observation, had gonorrhea which lasted six months, but was uncomplicated. Three years later there was stricture of the bulbous urethra. About ten months before coming under observation, a slight discharge occurred, which became chronic. There was a copious dribbling of urine after each micturition. At his first visit a 33 (F.) sound passed easily. Two months after the first visit, numerous papillomata were found in the anterior urethra by the endoscope. Nearly all these were removed by curetting, but some required repeated scraping. A few stumps which resisted the curette were cauterized with silver nitrate or a 50 per cent solution of chromic acid and so gradually removed. The discharge and the dribbling disappeared. Three years later, nothing suggesting papillomata could be found by the endoscope.

According to C. W. G. Rohrer,[2] chromic acid, though an excellent caustic, *is dangerous,* since toxic symptoms sometimes result and a few fatal cases are recorded. Intra-urethral papillomata, according to Rohrer, are best treated by the urethral forceps, the wire snare, the urethral curette or the galvanocautery. Hemorrhage may be controlled by direct application to the growths of adrenalin chlorid solution, 1:1000. The points from which the papillomata spring should be lightly cauterized with pure carbolic acid, glacial acetic acid or a little fused silver nitrate. Venereal

(1) Amer. Jour. of Derm., March, 1905.
(2) Amer. Jour. Med. Sciences, November, 1905.

ence at a stage of embryonic existence preceding the differentiation of the sexual glands into testicles and ovaries has given rise to the hypothesis that they are the cause of that differentiation.

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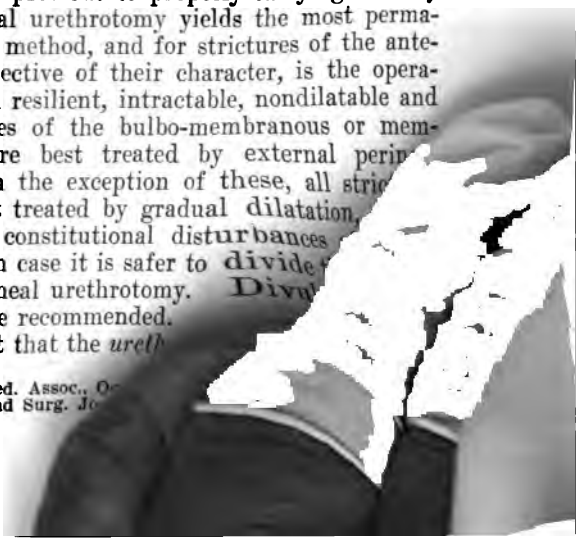

(1) Jour. Amer. Med. Assoc., O[ct.]
(2) Boston Med. and Surg. J[our.]

A new *varicocele operation* proposed by L. Lofton[1] is a modification of the ligature method. The scrotum is grasped with the left hand, the index finger and thumb separating the vas and spermatic artery, while the pampiniform plexus is brought up against the anterior and upper walls of the scrotum, and held firmly. A curved needle 3 inches in length, is threaded with No. 2 fiddle-string and the mass of veins is transfixed with it. It penetrates the inner scrotum wall, but does not perforate it. The needle is so guided as to slip along the inner wall, making a half circuit of the interior, above the transfixed vein, and brought out at the original puncture, where it is tied to the end of the external ligature. The mass of the veins should be drawn well up against the anterior wall of the scrotum. From one to two ligatures may be placed, as thought necessary. If the cord and artery have been separated from the general mass, the ligature can be tied firmly at once; if not, it should be gradually tightened so as to indicate the condition of affairs. Secondary inflammation binds the interior wall to the veins, bringing about a shortening thereby, securely anchoring them. The operation need not interrupt the patient's avocation. Severe inflammation does not follow the operation. In after-treatment, a well-fitting suspensory should be worn for some weeks.

Repair of vesical lesions in the rabbit has been studied by L. Francassini.[2] The lesions studied were puncture, suture after incision, ligature, cauterization, scraping and torsion of the mucosa. If puncture be done with a fine needle, repair of the lesion is too prompt to admit of study. If a $\frac{1}{2}$-inch diameter needle be used, recovery occurs in ten days, but vesical functions are unimpaired and histologic changes slight. Catgut suture, with the cut edges in contact, gives the best result. Lembert's suture endangers formation of a muscular spur without epithelium and liable to necrosis followed by urine infiltration. In accidental ligature, if not too tight, the ligature may become encapsulated. If tight, it may migrate to the bladder, to become a calculus nucleus. The bladder is singularly tolerant of cauterization and scraping, done delicately, and not too deep, is followed by complete restoration of the epi-

(1) N. Y. and Phila. Med. Jour., Nov. 19, 1904.
(2) Il Policlinico, March, 1905.

thelium about the beginning of the second week. The rate of restoration varies with the extent of abrasion. Repair occurs in fifteen days after compression or torsion of the epithelium alone. If muscle and epithelium necrosis result, new formation of connective tissue repairs the lesion completely in about 30 days.

H. M. Sherman[1] reports a case of *bladder exstrophy* in a boy, successfully treated by Peters' method. The ureters individually, keep a small circular patch of bladder wall about the vesical orifice of each, and ablation of the rest of the bladder wall. Each ureter with its button of bladder wall is then drawn through a small slit in the rectal wall and left hanging from .4 to .6 inch into the rectal lumen. Sherman varied from Peters' technic in not retaining a catheter in the ureter after transplantation, and also in using a suture at the rectal slits, including in the stitch a little of the peritoneal areolar tissue which had been kept with the ureter to carry its blood vessels. He thinks it might have been possible even to close up the abdomen at once had the patient been in better condition, as urinary leakage was impossible, but he followed Peters' method of temporarily packing the bladder wound. He left the prostatic urethra untouched, and later succeeded in permanently closing it above. The child was thus left with the rectum acting as a bladder and the genital passages intact. The functional results were good from the first. Ascending infection may, Sherman thinks, be indefinitely postponed, and the patient may go through life with comparatively little inconvenience and only an abdominal scar left as externally visible evidence of the operation. The results of R. Peterson's experiments on dogs do not justify Sherman's optimism as to ascending infection.

In *bladder exstrophy,* Muscatello[2] advises the following modification of Maydl's operation. A lateral anastomosis is made between the upper and lower ends of the sigmoid flexure, and the free loop severed at its upper end, which is closed, as is also the cut end of the sigmoid. This leaves an intestinal sac attached merely at its lower end. A piece of the bladder wall including the ureter mouth with at

(1) Jour. Amer. Med. Assoc., Sept. 25, 1905.
(2) Arch. f. klin. Chir., LXXXV, No. 4.

least .4 in. margin around them is next dissected free and sewed into a longitudinal incision in the upper part of the free intestinal sac and covered with peritoneum. The free end of this sac may be attached to the colon to prevent kinking. Long rubber tubes should be retained in the ureter during the operation. The abdominal wounds should not be closed entirely, drainage being left for about five days.

Seminal vesiculotomy has been performed by Fuller[1] in 33 cases for the treatment of seminal vesiculitis without a single death. Fuller claims, ignoring psychic impotence, that in the majority of instances where the male sexual function is crippled, the chief cause is a diseased condition of the seminal vesicles. After seminal vesiculotomy, the line of incision into the sac closes spontaneously, just as does the bladder incision in cystotomy. After the incision has so closed, the power to ejaculate returns.

Testicle ectopy, according to Lanz,[2] includes two types. In one is found every testicle that deviates from its normal path to the scrotum, and the other includes testicle retention where the organ is held somewhere in its normal descent. The last type is much more frequent. The claim of Curling that a testicle which does not descend within the first year of life remains permanently ectopic is shown to be erroneous by a case reported by Lanz in which the testicle first appeared at puberty in the external inguinal ring and finally reached the neck of the scrotum. The predisposing factor of ectopy, according to Lanz, is heredity. In 52 operations he failed to find the peritoneal adhesions which should result from recovered fetal peritonitis. The final descent in retention is never prevented by the vas deferens, although the shortness of the vessels occasionally interferes. The ectopic testicles as a rule exhibit marked macroscopic changes. They are small, flabby, often atrophy, and the still well developed epididymis is always lifted up from the testicle. The nerve and blood supply is not a cause, but arrest of development is probably the cause of the failure of the descent. Lanz castrated on one side 11 of his 51 cases because of marked atrophy and uncertainty of safe descent later. In but one case was active

(1) Post Graduate, October, 1904.
(2) Zentralblatt f. Chir., April 22, 1905.

spermatogenesis present. The albuginea as a rule is thickened; sometimes there was three to five times the normal thickness. Interstitial tissue was sometimes so much increased that the seminiferous tubules were scattered round as isolated islands. Atypic glandular epithelium sometimes was found, supporting Cohnheim's rôle of misplaced embryonal cells. The testicle has a special inclination to malignant degeneration.

Aid is usually sought on account of complications; the most frequent of which is hernia. Patients rarely complain of sexual disturbances. Lanz advises castration where there is marked testicle atrophy. It prevents the return of the condition frequently following orchidopexy, as well as later malignant degeneration. The remaining testicle assumes the duty of the removed organ. Of 14 cases treated two years before Lanz's paper, castration was done in 6. Children during the first decade of life were rarely subjected to operation. Massage was used instead; stroking along the inguinal canal and drawing down the testicle. In 8 of the 14 cases Lanz did what he calls "extension of the testicle." The vaginal process was cut and the upper portion treated as a hernial sac. The spermatic cord was then stretched carefully and the posterior wall of the canal reconstructed on the Bassini principle. With a long suture the testicle was caught near its lower pole and the suture passed through a buttonhole in the fundus of the often poorly developed scrotum. Under tension the suture was fastened with strips of plaster to the adjacent part of the thigh, or was bound to a transverse band fastened between the slightly spread thighs with plaster-of-paris bandages. The inguinal wound as well as the buttonhole was then closed.

Typhoid orchitis, according to D. Blumenfeld,[1] generally appears during convalescence, but much less frequently during the active course of the disease. Its onset is marked by a rapid rise of temperature, pain in the scrotum and occasional vomiting. The pain is sometimes rather slow in developing but usually increases rapidly in the neighborhood of the epididymis and vas; it may radiate into the loins and inguinal regions, intercostal spaces,

(1) British Medical Journal, Aug. 19, 1905.

and the thighs in front and behind. Typhoid orchitis unlike gonorrhea is usually unilateral. It ends in resolution or in suppuration (29 out of 69 cases); atrophy is the least common ending. The duration varies from twelve to eighteen days in the non-suppurating cases.

Testicle embryoma is reported by A. Müller[1] in a child of 2. There is but one instance on record where it occurred in an adult. Sometimes the embryoma is congenital, but it may develop very slowly in the course of a few months to twenty years. Malignant degeneration has never been observed. Embryoid testicle tumors are much more frequent, and as a rule occur after puberty. They generally grow rapidly and are sometimes first noticed after trauma. Malignant degeneration, especially sarcoma, is here not uncommon. The true embryoma with predominating ectodermal elements is much rarer.

Bladder sarcoma, according to J. A. Wilder,[2] is most common after middle age and during childhood, but may occur at any age. It is most frequent in males. The most common symptom is hematuria, but this is probably indicative of neoplasm ulceration rather than appearance. The next most common symptoms are cystitis and vesical irritation. Emaciation is present in advanced cases only. Calculus may complicate the condition. In women, the growth may invade the urethra and appear at the vaginal opening. The disease is more rapidly fatal in children than adults. Diagnosis has been made early where the neoplasm has been small, single and apparently localized. Its most common location is at the base of the bladder in the vicinity of the ureteral orifices. The neoplasm is usually sessile with a broad base, it is generally soft and pliable, more or less lobulated in some cases, presenting a cauliflower or villous appearance. The growths are usually single, but later may be multiple. Metastases are rare, except in quite advanced cases. The varieties which occur in the bladder are: Round-celled (large, medium, small, lymphosarcoma and alveolar), spindle-celled, mixed-celled, giant-celled, fibrosarcoma, myosarcoma, myxosarcoma and chondrosarcoma.

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(1) Arch. f. klin. Chir., B. XLIV.
(2) Amer. Jour. Med. Sciences, January, 1905.

Bladder chorio-epithelioma is described by W. S. Djewitzki[1] in a 75-year-old woman who died soon after curetting for fibroid hemorrhage. An ulcerated reddish mass occupied the greater part of the bladder. Numerous metastases were found in the lungs, intestines and spleen. The tumor and its metastases were made up of Langhans cells and syncytium. The tumor may have developed from the Wolffian duct normally present in the posterior bladder wall during embryonic life or may have begun in the epithelial bladder lining.

Soft rubber catheters are, according to Krotoszyner,[2] rendered sterile by being boiled five minutes in sodium chlorid solution, which should fill the lumen of the catheter. They should be washed with soap spirits and running water after use. Hard rubber, silk, and cotton woven catheters should be boiled five minutes in a saturated solution of ammonia sulphate. Each instrument should be wrapped separately in gauze or a towel, or if several are to be sterilized, in such a manner that their surfaces will not come in contact with the sides of the vessel or other catheters. Ureter catheters can be folded and wrapped in a towel so their surfaces are kept apart, and boiled for five minutes in a saturated solution of ammonium sulphate. Cystoscopes should be sterilized by first washing them in soap spirits and water, then vigorously rubbing them for two minutes with two different pieces of gauze or cotton, wet with soap spirits and then with alcohol for one minute. The channel for the urethra catheter can be cleansed by a brush like that used for pipe cleaning, first brushing with soap spirits and then with alcohol. Instruments can be kept aseptic if they are snugly wrapped in gauze or towel, wet with soap spirits.

The majority of cases of *chronic prostatitis* are the result of gonorrheal infection plus pelvic congestion, according to E. G. Ballenger.[3] Congestion may be due to traumatism from instrumentation, etc., prolonged ungratified sexual desire, masturbation, excessive venery, constipation, hemorrhoids, jarring of the perineum, irritating applications, strongly acid or alkaline urine, urethral cal-

(1) Virchow's Archiv, CLXXVIII.
(2) Med. News, Aug. 27, 1904.
(3) Ibid., May 10, 1905.

culi, prostatic plexus varicosities, bladder over-distension, atheromatous vessels, chilling and over-fatigue. Catarrhal inflammation may occur from congestion alone. There is then no pus but a discharge of clear glairy mucus, or mucus and phosphates.

Transvesical prostatectomy, according to Nicolich,[1] is preferable to the treatment of prostatic hypertrophy with retention by antiseptic successive evacuation of small quantities of urine. Of 10 cases treated in the last fashion 9 died; while in 4 cases treated by transvesical prostatectomy there were 4 recoveries. All four patients had been bed ridden and had suffered severely with incontinence accompanied with enormous retention; one was in a cachectic state and one had cardiac disease. All recovered without the complications generally associated with catheterization. After from seventeen to thirty days urination was spontaneous.

The *prostate,* according to P. M. Pilcher,[2] *consists essentially of two lateral lobes* connected together in front of the urethra by the anterior commissure and beneath the urethra by the posterior commissure. The prostatic urethra traverses the gland a little in front of its median line from base to apex. The cortex consists mainly of unstriped muscular fiber slightly mixed with fibrous tissue. This constitutes the "capsule" of the prostate. Outside is an indefinite sheet of fibrous tissue in which is located the prostatic venous plexus. The organ consists of stroma and glandular elements in variable proportions. The gland contains from 40 to 60 lobules of alveolar type. Prostates which require removal are, according to Pilcher, of three types: One is greatly enlarged but soft. The second is relatively small, contracted and hard. The third is of mixed type. All three types occur in gonorrheics. The urethra is distorted acording to the size, direction and extent of the hypertrophy. An atrophic form is not uncommon. The average weight in 23 cases operated on was about 250 grains, nearly the average weight of normal prostates. In the hypertrophied prostate there is both relative and absolute increase in glandular tissue. All degrees of change from the normal alveoli (cyst forma-

(1) Ann. des Mal. des Organes Genitourin, May, 1905.
(2) Annals of Surg., April, 1905.

tions and large adenomas) occur. In atrophy two types are produced; in the first, the glandular elements are decreased and smaller than normal. In the second type there is predominance of compressed glands with dilated hypertrophied lobules. Hypertrophy is due more to glandular overgrowth distorted and increased by senile degenerative changes than to any extrinsic inflammatory agency. The contracted type is distinct from the large soft type. In many hypertrophies there is true muscular hypertrophy. In some atrophies the glandular elements are diminished and the muscular, relatively increased. Hypertrophy of the prostate results from glandular overgrowth, influenced by senile degenerative changes, and other states which tend to increase fibrous connective tissue in an actively functionating organ.

Prostate carcinoma, according to H. H. Young,[1] occurs in about 10 per cent of the cases of prostatic enlargement. It may begin as an isolated nodule in an otherwise benign hypertrophy, or a prostatic enlargement which has for many years furnished the symptoms and signs of benign hypertrophy may suddenly become clearly malignant. Marked induration, if only an intralobar nodule in one or both lobes of the prostate in men past 50 should be viewed with suspicion, especially if the cystoscope shows little intravesical prostatic outgrowth, and pain and tenderness are present. The posterior surface of the prostate should be exposed as for an ordinary prostatectomy, and if the operator is unable to make a positive diagnosis of malignancy, longitudinal incisions should be made on each side of the urethra (as in prostatectomy) and a piece of tissue excised for frozen sections, which can be prepared in about six minutes and examined by the operator at once. If the disease be malignant, the incisions may be cauterized and closed and the radical operation performed. Prostate cancer remains within the confines of the lobes, the urethra, bladder and especially the posterior capsule of the prostate resting inviolate for a considerable period. Extraprostatic invasion nearly always occurs first along the ejaculatory ducts into the space immediately above the prostate between the seminal

(1) Johns Hopkins Hosp. Bull., October, 1905.

vesicles and the bladder and beneath the fascia of Denonvilliers. Thence the disease gradually invades the inferior surface of the trigone and the lymphatics leading toward the lateral walls of the pelvis, but involvement of the pelvic glands occurs late and often the disease metastasises into the osseous system without first invading the glands. Cure can be expected only from radical measures and removal of the seminal vesicles, vasa deferentia and most of the vesical trigone with the entire prostate.

Three methods of *prostate operation* are recommended by Willy Meyer.[1] The adoption of an exclusive method is unwise. Perineal, and suprapubic prostatectomy, and galvano-caustic prostatotomy has each its own decided merits, and holds a distinct place in surgery. Operation for enlarged prostate must be urgently recommended to most patients with enlarged prostate as soon as it becomes necessary for them to use the catheter habitually. The death rate following operations is 5 per cent or less; the mortality due to pyelonephritis resulting from self-catheterization is much greater. Meyer has performed Bottini's operation on 59 cases, perineal prostatectomy 9 times, the suprapubic operation 22 times. The only deaths by the perineal method were from the anesthetic in one case, and in a case almost moribund at the time of operation. Of the 22 suprapubic operations, 17 cases of benign hypertrophy are living, 2 died of causes not due to the operation; 3 of cancerous hypertrophy died of causes due to the nature of the disease. Meyer believes it possible to cut the grooves with the galvano-cautery both deep and wide; that a median lobe is a contraindication to the Bottini operation, if it is possible to do an enucleation; the cystoscope is of value in explaining the conditions at the neck; Bottini's operation, even when done twice, does not prevent a prostatectomy, should that become necessary. But, being a purely intravesical operation, it has many failures, and sometimes entails tedious after-treatment. If radical operation is refused it should be done. When prostatectomy is done full power over the urine is obtained. A small portion of the urethral mucous membrane may have to be removed with the gland, but this

(1) Medical Record, Nov. 3, 1905.

is no disadvantage. There is little choice between the perineal and suprapubic routes. In the perineal operation there is a rapid return to normal control of the urine, and leakage over the abdomen is not present. The suprapubic operation can be done in less time. Cystoscopy should always be performed before Bottini's operation, as the hypertrophy may be entirely intravesical. In advanced carcinoma, Bottini's operation is to be preferred, since all cancerous metastases cannot be removed. A gland palpable by rectum and rising not far from the sphincter muscle can best be attacked from below; when higher up and projecting into the bladder it should be operated on from above. When soft and composed of small lobes, operate from below. When complicated by a large calculus work from above. The preservation of sexual power is important, and the suprapubic method retains it in the largest number of cases.

Urethral stricture *accompanied with complete retention and a calculus impacted* in the stricture is reported by J. W. Dowden.[1] The man had been treated for stricture 11 years previous to the report; six months before admission, dysuria and frequency of micturition occurred. The evening before admission, there was complete retention. The bladder was distended the next morning. There was but little prostatic enlargement and an instrument could not be passed further than the membranous urethra. The following day a little urine escaped. External urethrotomy was performed, but on inspection of the stricture area, the proximal opening could not be discovered. The dense fibrous stricture tissue was cut through in the middle line. Near where the upper urethra wall should have been was seen a tiny reddish yellow spot. This was found to be gritty and hard and was picked out, showing the urethral orifice with characteristic smooth bluish white membrane. A probe was easily passed into the bladder, the stricture divided and a full size instrument tied in. The gritty nodule was a minute uric acid calculus of pinhead size.

An enormous urethral calculus is reported by C. D. Fisher[2] in a 49-year-old cattleman who had lived for years

(1) Scottish Med. and Surg. Jour., March, 1905.
(2) Amer. Med., Oct. 21, 1905.

in the saddle. At ten the urethra was obstructed by a growth and ineffectual efforts were made to pass a sound. A urinary fistula formed behind the growth which was the only way that the patient was able to empty his bladder for years. On longitudinal incision a stone was found in a greatly extended urethra completely obstructing it from the glans to the bulbous portion in the perineum. After removal of the stone the urethra was cut down to its normal size and a catheter was passed into the bladder, perineal drainage was established, the wound closed and a moist dressing applied. In a week the perineal drain was removed and a catheter passed the full length of the urethra. The patient was subsequently discharged completely recovered. The diameters of the calculus were $2\frac{3}{8}$ inches and $5\frac{1}{2}$ inches; the circumferences were $7\frac{1}{4}$ inches and 13 inches; the weight was $19\frac{1}{2}$ ounces.

Transperitoneal uretero-lithotomy is reported by J. W. Bovee[1] in the case of a ureteral calculus which weighed 1,310 grains. It was kidney-shaped (one end larger than the other), grayish in color and rough on the surface. It consisted of several concentric layers of phosphatic nature covering a decidedly dark mass an inch in diameter formed of layers of calcium oxalate.

Pathologic desire to urinate and female cystitis have been found frequently associated by R. Knorr.[2] One out of five of gynecologic patients complain of disturbances in urination, and one out of every eight had chronic cystitis of the bladder neck. The cystitis may be due to infection or to venous congestion.

Bladder catarrh in early pulmonary tuberculosis is quite frequent according to Campani.[3] He claims this catarrhal condition is due to lesser acidity of the urine in the pretuberculous stage and in incipient tuberculosis. Hypoacidity favors development of pseudo-phosphaturia and cystitis and is depressing to the organism in general.

Micturition difficulties in childhood are often due, according to A. Mousseau,[4] to hyperacidity and gravel which in the sensitive bladder of the child excite micturition before vesical distension occurs. In the new-born the first

(1) Washington Med. Annals, September, 1905.
(2) Zeitschr. f. Geb. und Gynak. B. XLIV.
(3) Gaz. degli Ospedali, June, 1905.
(4) Rev. des Mal. de l'Enfance, May, 1905.

urine is charged with urates and uric acid consequent upon elimination of renal uratic infarcts. The first micturition may be delayed three days after birth without bladder distension but with evidence of pain. Painful spasms of the bladder neck occur which renders micturition difficult but cease when the urine increases in quantity. In nurslings micturition accompanied by crying is often due to uric acid-sand irritation. Under two years certain children suffer from dysuria without vesical lesion where there is diminution and concentration of the urine. This condition is frequent where there is insufficient oxidation of nitrogenous substances eliminated by the kidneys.

Camphoric acid as a *preventive of catheter fever* is recommended by Dafreudenburg[1] in 15-grain doses once, twice or three times daily in capsules, beginning a few days before catheterization and continuing a couple of days afterwards. It exercises a calmative influence on cystitis. It is contraindicated by gastric disturbance.

Toxemic paraplegia cured by internal urethrotomy is reported by R. de Grau.[2] The patient, a 50-year-old man, had in youth a very persistent gonorrhea. For about five years before the operation there was difficulty in urination. The jet had not its normal impulse, and at times there was fairly complete retention. Sometimes thereafter the patient found his legs growing so weak that he could not stand for any length of time. Tactile sensibility of the feet was diminished. Heat and pain sense was normal. Muscular power in the legs was greatly diminished. The knee-jerk was normal but the plantar reflex was diminished. After internal urethrotomy all the symptoms disappeared rapidly.

Cystic ureteritis, according to E. Simelew,[3] is determined by chronic catarrhal processes affecting the ureteric mucosa. The cysts originate in the crypts of von Brunn. Epithelium lines the sac. The cysts may conglomerate causing multiple formations. The von Brunn structures are sometimes absent. Diminution of the ureter calibre may play a part in cyst etiology.

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(1) Gaz. degli Ospedali e delle Clin., 1904, No. 38.
(2) Arch. de Terap. de las Enfermed. Nerv. Ment., January, 1905.
(3) Il Policlinico, August, 1905.

NERVOUS AND MENTAL DISEASES

BY

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NERVOUS DISEASES.

THE NEURONE THEORY.

During the last year attention has been chiefly directed by investigators toward the neurofibrillæ described by Apathy and by Ramon y Cajal and others. Bethe,[1] in an article concerning the present state of the neurone theory, defends his position of antagonism. He admits that pathology and physiology have done much to advance Waldeyer's morphologic doctrine of the individuality of the neurone. Forel's doctrine of the trophic unity of a neurone, based upon the facts of both Wallerian and retrograde degeneration has also afforded strong evidence in favor of the neurone doctrine. As Bethe sees it the neurone theory in its original form declares: (1) The neurone is an embryologic unit, originating from a single embryonic cell. (2) The neurone is a morphologic unit, even in the adult stage. (3) There are no nervous elements other than neurones. (4) The neurones are merely in contact with one another, the branches of dendrites and the collaterals and end-apparatuses of neurones always ending blindly. (5) The neurone is a trophic unit. (6) The neurone is a functional unit. At the present day, according to Bethe, very few investigators will unqualifiedly admit the truth of all six propositions. The third proposition, that there are no nervous elements other than neurones, is most severely attacked. Moreover, Bethe does not find there is good evidence that the neurone theory applies to all grades of nervous systems, including those of the lowest form of life. For example, in medusæ and polyps, and in the sympathetic vascular plexuses, it has been shown by Golgi and Dogiel that there is no differentiation of cell-processes into axones and dendrites, but that they form a true network; and according to the observations of Apathy, Bethe, Holmgren, Wolff and others, similar networks occur in all

(1) Deutsche medizinische Wochenschrift, Aug. 11, 1904.

classes of the animal kingdom. Moreover, those fibres which in these cases pass into the musculature, do not issue from a single ganglion-cell, but approximately from the center of a fibre connecting two ganglion-cells. If anything is an axone, this motor fiber is one; yet it issues from *two cells.*

Embryologically the neurone theory is not impregnable. Despite the assertions of His, other observers, notably Balfour, Beard and Dohrn, believe that nerve-fibers develop from chains of cells. Certainly the motor and sensory neurones of the vertebrate nervous system are not individual cells. Though Golgi's method and the methylen-blue stain indicate the embryologic unity of nerve-cells, the use of other methods has led to the discovery of pictures difficult to harmonize with the neurone theory. While the neurofibrillæ pass without a break through the nodes of Ranvier, nevertheless, according to Mann, Monckberg and Bethe, plates intervene at those points; and these plates mean cell-boundaries, says Bethe. He therefore concludes that a neurone is a multicellular formation.

The neurofibrillæ, found by Apathy, do not remain confined within the neurone. In many instances they may be seen to form networks, or pass straight from one neurone into another. Concerning Ramon y Cajal's recent work, mentioned in the Practical Medicine Series of September, 1904, Bethe affirms: "The recently published claim of Ramon y Cajal's that no fibrillary networks intervene between the neurones of Hirudineæ is very certainly, at least according to the figures, based upon imperfect stains." The neurofibrillæ are undoubtedly cell-products, but it is not always possible to point out the mother-cell. They seem to be nerve-elements rather than parts of neurones, and hence the neurones are not the only nerve-elements existing.

Pathologically, the laws of Waller and Gudden do not support the neurone hypothesis. Again, in the case of regenerations, as shown by Howell and Huber, the cells of Schwann's sheath take a very active part. Moreover, autoregenerative processes have been described by Bethe himself, van Gehuchten and others. Such facts seem to Bethe to indicate positively that the neurone is not a trophic unit. Neither is the neurone a single cellular element since the axone regenerates without any connection

with the cell-body. The axone, therefore, is much more like a cell-colony than a single cell. That injuries to a neurone never spread beyond its confines is also not true. A large number of observations indicate that in neighboring ganglion-cells slight changes occur. If the glossopharyngeal nerve be cut, for example, the taste-cells entirely disappear in three weeks' time, and, according to the neurone theory, these taste-cells are peripheral sensory neurones.

Finally, the neurone is not a physiologic unit. For example, in one of the arthropods the ganglion-cells may be entirely removed, and, provided the cell-processes are not injured, the reflex phenomena still remain present. Bethe concludes as follows: (1) "The neurone cannot be regarded as a cellular unit, because the later embryologic and histologic findings and the facts of autogenous regeneration prove that the axis-cylinders of the peripheral nerves, at least, are of multicellular origin. (2) It is very probable that in addition to neurone complexes there are other nerve-elements which are genetically independent of the former. (3) Nerve-cells in both vertebrates and invertebrates are connected with one another by broad anastomoses, constituting a network. (4) The neurone represents neither a trophic nor a functional unit."

Lugaro,[1] writing upon the present state of the neurone theory, concludes as follows: (1) The neurone can be disputed as a cellular unity, but it is surely an anatomic unit. The demonstration of neurofibrillæ and of their passage from one neurone to another does not destroy the conception of the anatomic individuality of a neurone. (2) This continuity has not been demonstrated in vertebrates; it is probable that such continuity among invertebrates is of a special sort, a kind of adaptation to a method of functioning not found among vertebrates. (3) The law of dynamic polarity which assigns to the cellular bodies and the dendrites a receiving function, and to the axones a discharging function, in its general outlines, is still intact; in some particular cases this law may be modified by the passage of neurofibrils or by the formation of the fibrillary network. (4) The Wallerian law, even though the multicellular origin of the axone and the autogenous regenera-

(1) Archiv. de Anatomia e de Embrol., III, pp. 412-437.

tion of the peripheral nerves be accepted as demonstrated facts, still stands as the law of trophic control in the adult organism. [These conclusions of Lugaro, who is eminently qualified to write upon the subject, doubtless represent the best opinion concerning the neurone hypothesis. Bethe's views, at first bitterly resisted by the many, have been slowly gaining ground and have led to a partial modification of the original dicta concerning the neurone first uttered by Waldeyer in 1891.—ED.]

SYMPTOMATOLOGY.

Kernig's Sign. Sainton and Voisin[1] published during the past year a general review of Kernig's sign, beginning with an account of its history since 1883, when it was first presented by him at the medical society of St. Petersburg. The sign, usually searched for in the lower extremities, is also met with in the arms. It may be obtained with the patient in the sitting posture, and almost equally well when he is recumbent. In the latter case the examiner must be sure that the bed is firm and flat, else the hips may sink down in the middle; and in the former case he must be sure that the patient is comfortable. The degree of flexion at the knee-joint is variable; if the sign is very marked it does not exceed a right angle, but this is rare. As a rule the angle does not exceed 135 degrees, and one author (Shields) does not admit that it exceeds 120 degrees. Kernig's sign in the upper extremities is far from constant, and does not have the symptomatic value which it possesses in the lower extremities; hence search for it is frequently neglected.

The diagnostic value of Kernig's sign is indicated by the fact that it has been found present in more than three-quarters of the cases of meningitis, whether due to various bacteria, as the diplococcus of Weichselbaum, pneumococci, typhoid bacilli, and others, or due to the tubercle bacilli. It has no value whatever in differentiating the various forms of meningitis. Interesting are those cases in which Kernig's sign is associated with a disap-

(1) Gazette des Hôpitaux, August 27, 1904. (A very extensive bibliography is appended.)

pearance of the knee-jerk. It also occurs in meningeal hemorrhage, and has led to a proper diagnosis as proved by lumbar puncture. It has been found in 11.8 per cent of cases of typhoid fever, generally in the grave cases only. For example, if the mortality of ordinary cases is 7 per cent, the mortality among cases in which Kernig's sign is present is 25 per cent, and if among average cases of typhoid fever 16 per cent show relapses, of those presenting Kernig's sign 43 per cent show relapses. Next to typhoid fever pneumonia among the infectious diseases presents the greatest number of cases. It appears as early as the fourth day, sometimes isolated, and sometimes accompanied by delirium, headache or oculo-pupillary disturbances. Yet in these cases lumbar puncture shows the cerebrospinal fluid to be clear. It has been found in empyema, and the gastrointestinal infections of very early childhood. Among the intoxications, uremia is about the only one in which it has been found, except possibly during the resorption of certain edemata.

Aside from the above-mentioned affections and intoxications, Kernig's sign has been observed almost exclusively in such affections of the nervous system as are painful or spastic. It has been found in cerebellar hemorrhage, in cerebral hemorrhage, in old hemiplegics, in cerebral abscess, in meningo-myelitis of syphilitic origin, in tabes dorsalis, especially at the crises, in lumbago and sciatica and even in the meningism of hysteria.

The authors further discuss the nature of Kernig's sign and its pathogenesis. It is undoubtedly merely a contracture of the flexor muscles of the leg, existing normally but much exaggerated in disease. The theory that it is due to hypertension of the cerebrospinal fluid is untenable since it occurs in some instances when lumbar puncture fails to show any increased tension whatever. The theory of meningo-medullary irritation has been advanced in three different forms: (a) The theory of irritation of the spinal cord and of the cauda equina; (b) the theory of irritation of the pyramidal tract; (c) the theory of irritation of the cells of the anterior horn of the spinal cord. Of these the latter is the more preferable, but none meets all conditions of the presence of the sign. The theory that the sign is due merely to a reflex from pain is untenable because the

sign persists when pain is annihilated by cocain, as observed by Abadie in a case of sciatica.

The conception which seems most simple to the authors makes of the sign a reflex phenomenon, caused by the irritation of the excito-reflex cells of the cord by various influences. Thus it is easy to explain its presence when associated with peripheral disease, as sciatica and lumbago; in hemiplegics it would indicate such an irritation; in meningitis it might depend upon local meningeal irritation of either exogenous or endogenous origin.

Ankle-Clonus. Gilbert Ballet[1] two years ago reported a case at a meeting of the Neurological Society of Paris, in which headache and amyasthenia were the only symptoms, with the exception of an intermittent clonus of the right foot. At that meeting (February 5, 1903) he could do nothing more than make a diagnosis of neurasthenia, and there arose a discussion as to whether ankle-clonus might not occur in hysteria and neurasthenia, some present taking the affirmative side. Since that time new symptoms occurred, such as lymphocytosis of the cerebrospinal fluid on lumbar puncture, increased headache and vomiting. A diagnosis of meningitis of slow evolution was then made instead of neurasthenia. Later ankle-clonus appeared intermittently in the left foot as well as the right, associated with trembling of the legs and of the tongue. Death having followed January 1, 1904, a postmortem examination was made and showed an evident thickening of the pia mater at the base of the cerebral hemispheres, beneath the pons and medulla, and along the cerebral peduncles in the interpeduncular space. This thickening was made up of layers of fibrin and nuclear débris, and was due to syphilis. No lesion of the pyramidal tract was found by staining of sections according to the methods of Pal, Marchi and Van Gieson; and examination of the pyramidal cortical cells in the region of the fissure of Rolando by Nissl's method gave negative findings.

Evidently, therefore, in this case the ankle-clonus did not depend upon a *lesion* of the pyramidal tract, but seems to have been due to irritation of the pyramidal tract fibers during their passage through the cerebral peduncles.

(1) Revue Neurologique, 1905, p. 132.

"There is, then, not only a clonus due to an *alteration* of pyramidal tract fibers, but also a clonus due to simple *irritation* of such fibers." Possibly clonus from alteration is permanent, or at least habitually present, associated with increased knee-jerks and Babinski's toe-sign; whereas, clonus from irritation is intermittent, possibly at first unilateral, without increased reflexes or Babinski's toe-sign. In the discussion following Ballet's presentation of his case, Babinski declared that in his opinion ankle-clonus never occurs in hysteria or neurasthenia.

Lannois and Clement[1] in a paper on ankle-clonus during anesthesia reported at the meeting of the Paris Neurological Society of May 11, 1905, state that ankle-clonus, not present before anesthesia, comes on from four to seven minutes after cutaneous anesthesia, and only in very complete anesthesia. It persists even after the reappearance of the normal ocular reflex. This ankle-clonus is independent of muscular tonicity, since it is produced during atony and complete flaccidity of the muscles. Clinically ankle-clonus and increased knee-jerk are frequently intimately related, but in anesthesia, ankle-clonus, not originally present, appears and increases in intensity, whereas the knee-jerks entirely disappear. Contrary to current opinion there seems to be no relation between increased reflexes and exaggeration of muscular tonus. The authors draw the following conclusions: (1) "During anesthesia there is at first a disappearance of the cutaneous and ocular reflexes. The knee-jerks on the contrary are exaggerated before they disappear. (2) Ankle-clonus does not behave as does the knee-jerk; it begins to increase a few instants after the disappearance of ocular reflex action, but instead of diminishing during the further course of anesthesia it goes on increasing and persists even when the patient has awakened. (3) This exaggerated ankle-clonus is completely independent of the degree of muscular tonus. It appears to have its maximum during complete muscular relaxation. (4) The clonus center is probably intermediate between the centers for the tendinous reflexes, and the centers for the organic reflexes of circulation and respiration; hence the importance of watching ankle-clonus during anesthesia.

(1) Revue Neurologique, May 30, 1905,

The Plantar Reflex and its Modifications in Disease. A rather voluminous literature sprang up during the year just passed, based upon the group of plantar reflexes. Verger and Abadie[1] split up the toe-sign of Babinski into three subdivisions, the plantar-digital, the plantar-tibial and the plantar-crural, depending upon the location of the muscles which are called into activity by irritation of the sole of the foot. An attempt was made of a graphic study of these reflexes in the normal state and in some spastic affections involving the pyramidal system. A complicated set of myographs was devised and all obstacles overcome. The plantar-crural reflex showed a regularly arched curve, the plantar-tibial reflex showed several smaller curves corresponding to the different muscle contractions, and the plantar-digital reflex showed two chief types: (1) An almost perpendicular up stroke with a sharp apex and a steep down stroke, corresponding to flexion of the great toe in health; (2) a reversed letter S, of which the upper loop is larger than the lower, and of which the lower loop passes below the base-line, gradually turning upward to it at the end. This latter curve follows the flexion of the great toe, and is really a succeeding extension; it is to be found only by means of the myograph. Thirty cases of fresh and old hemiplegics, Pott's disease, multiple sclerosis and transverse myelitis were studied. The plantar-crural and plantar-digital reflexes were approximately normal, except that the degree of oscillation was greater. In patients with clearly cut spastic symptoms, flexion only of the great toe was found. Interesting is the fact that the myograph did not yield identical pictures in identical lesions.

Bertolotti[2] discusses the presence of Babinski's toe-sign in extra-pyramidal lesions, and its genesis. He found the sign present in two cases which showed no lesions of the pyramidal tract; the first, a man of 50 years, with paralysis of the upper roots of the sacral plexus following Pott's disease; the second, a child of 5 years, with acute anterior poliomyelitis. In each case Babinski's toe-sign was present, probably because of the disturbance of the normal antagonism existing between extensors and flexors; yet

(1) Nouvelle Iconographie de la Salpêtrière, XVII, p. 67.
(2) Rivista di Patalog. nerv. e ment., IX, 1904. pp. 430-448.

there was no lesion of the pyramidal tract. Probably there exist, independently of the corticospinal tracts, other pathways, less well differentiated, of a cortico-mesencephalic-spinal course which send impulses to the extensor group of muscles only, and after injury or destruction of the pyramidal tract these other pathways act vicariously. Thus it is with the Babinski reflex, with the Oppenheim reflex, with the tibial sign, and in part with the femoral reflex of Remak. Doubtless the plantar reflex and the Babinski reflex both start from the same center, but one set of impulses comes down the pyramidal and the other down the extra-pyramidal pathway. Muscles which have no antagonists, as the abdominal and cremasteric, therefore show no pathologic reflexes.

Stanley Barnes[1] in an article on the diagnostic value of the plantar reflex thinks that any lack of unanimity among authorities as to the diagnostic meaning of the sign is due to lack of exact observation or exact description of facts observed. In healthy adults Babinski's phenomenon never occurs; when it is present it always means organic disease, though a visible lesion of the pyramidal tracts is not necessarily present. Strong increase of intracranial pressure, for example, from meningitis or from tumors leading to comatose states, may so disturb conductivity in the pyramidal tracts as to bring out the toe-sign. Purely functional conditions, as hysteria, or organic lesions not affecting the pyramidal tracts, as tabes, never show the Babinski sign. In hemiplegics the sign is, as a rule, absent upon the sound side, in spite of increase in the knee-jerks. Difficulties may be met with in what Barnes calls "pyramidal equilibrium." In this condition it is in the examiner's power by modifications of the method of obtaining the reflex to make either the extensor or the flexor element predominate. In such states the extensor reflex will predominate provided the following points be kept well in mind: Complete extension of the lower extremity, with relaxation of the limb, distraction of the patient's attention, a warm and dry footsole, and *slight* irritation along the external edge of the border of the foot from behind forward. The *first* response is the one upon which stress is to be laid; only confusion follows several attempts to

(1) Rev. of Neurol. and Psychol., May, 1904.

obtain the sign, for some responses will be by the flexors and some by the extensors. The threshold of the "pyramidal equilibrium" lies deeper in fresh, acute diseases of the pyramidal tracts than in chronic cases. Hence, in the former fewer pyramidal fibers need to be involved to give the reflex than in chronic cases. The value of the Babinski reflex (as compared with the knee-jerk) lies in its qualitative character.

Pfeiffer[1] investigated in Oppenheim's polyclinic seventy cases of diseases of the central nervous system with spastic symptoms, with reference to the significance of Babinski's toe-sign and of Oppenheim's calf reflex as an indication of pyramidal tract disease. Both signs were present thirty-eight times; one or the other was present twenty-one times; neither was present eleven times. In hemiplegics, in whom during the stage of paralysis these reflexes were present, the reflex became normal when the normal function was restored. From the point of view of diagnostic value, Oppenheim's reflex is the more valuable. In many cases of pyramidal tract disease only one of the two reflexes is present; hence, each must be looked for invariably. The pathways traversed by the extensor impulses, when the pyramidal tracts are blocked, are doubtless Monakow's bundle or the anterolateral tracts.

Crocq[2] discusses the combined plantar-reflex phenomena with special reference to *hysteria*. He enumerates the following reflexes as normal: (1) The plantar flexion reflex which he names the normal plantar reflex of Babinski; (2) the reflex of the fascia lata first described by Brissaud, and which Crocq names the normal plantar reflex of Brissaud; (3) the plantar defensive reflex, consisting of a contraction of the sartorius, adductors and anterior tibial muscles, with flexion of the foot on the leg, of the leg on the thigh, and of the thigh on the pelvis. The author enumerates the following reflexes as pathologic: (1) The dorsal flexion of the great toe, or Babinski's reflex; (2) and the adduction of the toes or fan-sign. His conclusions follow: (1) "Abolition of the pharyngeal reflex is frequent in hysteria (73 per cent); it is especially noteworthy in the forms accompanied by anesthesia (81.8 per cent); it

(1) Monatsschr. f. Psych. u. Neurol., XVI, p. 565.
(2) Revue Neurologique. November, 1904.

is present less frequently in the paroxysmal forms (74.6 per cent); it is still less frequent in hysterical paralyses and contractures (65.4 per cent). This phenomenon is met with in a large number of other affections, and even in health; it is therefore in itself not sufficient for establishing a diagnosis of hysteria. (2) Exaggeration of the tendon reflexes (79 per cent), more frequent in abolition of the pharyngeal reflex, constitutes a sign about as valuable as the latter for establishing a diagnosis; very frequent in the paroxysmal forms (84.1 per cent), it is less constant in paralyses and contractures (73.1 per cent), and still less in anesthesias (63.6 per cent). Its presence in a large number of toxic and infectious diseases, and even in health, robs it of any pathognomonic value. (3) The loss of plantar sensibility is frequent in hysteria (42 per cent); it is met with especially in forms accompanied by anesthesia (63.6 per cent); next in order of frequency are the paralyses and contractures (61.5 per cent); and lastly the paroxysmal forms (31.7 per cent). (4) The simultaneous abolition of the cortical plantar reflex or the flexor reflex, and of the medullary plantar reflex or reflex of the fascia lata, is very frequent (59 per cent); it is especially marked in forms with anesthesia (72.7 per cent), and is met with almost as frequently in forms showing paralyses and contractures (53.7 per cent) and in the paroxysmal forms (57.1 per cent). He gives to the simultaneous loss of both these reflexes the name *combined plantar phenomenon.* These reflexes being remarkably constant in the normal state, their abolition is of great importance in the diagnosis of hysteria. Therefore he believes we are justified in considering the combined plantar phenomenon as a special sign of hysteria. (5) Plantar anesthesia is not a condition *sine qua non* for the existence of the combined plantar phenomenon. It exists frequently (57.6 per cent) with the phenomenon, but the latter may be present without alteration of sensibility (20.4 per cent), just as plantar anesthesia may exist without alteration of the reflexes (13.6 per cent). (6) The deep plantar reflex is often exaggerated in hysteria (50 per cent); sometimes it is normal (24 per cent), diminished (16 per cent), or even absent (10 per cent). Its modifications are not parallel with those of the two other normal plantar reflexes. Though

classes of the animal kingdom. Moreover, those fibres which in these cases pass into the musculature, do not issue from a single ganglion-cell, but approximately from the center of a fibre connecting two ganglion-cells. If anything is an axone, this motor fiber is one; yet it issues from *two cells.*

Embryologically the neurone theory is not impregnable. Despite the assertions of His, other observers, notably Balfour, Beard and Dohrn, believe that nerve-fibers develop from chains of cells. Certainly the motor and sensory neurones of the vertebrate nervous system are not individual cells. Though Golgi's method and the methylen-blue stain indicate the embryologic unity of nerve-cells, the use of other methods has led to the discovery of pictures difficult to harmonize with the neurone theory. While the neurofibrillæ pass without a break through the nodes of Ranvier, nevertheless, according to Mann, Monckberg and Bethe, plates intervene at those points; and these plates mean cell-boundaries, says Bethe. He therefore concludes that a neurone is a multicellular formation.

The neurofibrillæ, found by Apathy, do not remain confined within the neurone. In many instances they may be seen to form networks, or pass straight from one neurone into another. Concerning Ramon y Cajal's recent work, mentioned in the Practical Medicine Series of September, 1904, Bethe affirms: "The recently published claim of Ramon y Cajal's that no fibrillary networks intervene between the neurones of Hirudineæ is very certainly, at least according to the figures, based upon imperfect stains." The neurofibrillæ are undoubtedly cell-products, but it is not always possible to point out the mother-cell. They seem to be nerve-elements rather than parts of neurones, and hence the neurones are not the only nerve-elements existing.

Pathologically, the laws of Waller and Gudden do not support the neurone hypothesis. Again, in the case of regenerations, as shown by Howell and Huber, the cells of Schwann's sheath take a very active part. Moreover, autoregenerative processes have been described by Bethe himself, van Gehuchten and others. Such facts seem to Bethe to indicate positively that the neurone is not a trophic unit. Neither is the neurone a single cellular element since the axone regenerates without any connection

with the cell-body. The axone, therefore, is much more like a cell-colony than a single cell. That injuries to a neurone never spread beyond its confines is also not true. A large number of observations indicate that in neighboring ganglion-cells slight changes occur. If the glossopharyngeal nerve be cut, for example, the taste-cells entirely disappear in three weeks' time, and, according to the neurone theory, these taste-cells are peripheral sensory neurones.

Finally, the neurone is not a physiologic unit. For example, in one of the arthropods the ganglion-cells may be entirely removed, and, provided the cell-processes are not injured, the reflex phenomena still remain present. Bethe concludes as follows: (1) "The neurone cannot be regarded as a cellular unit, because the later embryologic and histologic findings and the facts of autogenous regeneration prove that the axis-cylinders of the peripheral nerves, at least, are of multicellular origin. (2) It is very probable that in addition to neurone complexes there are other nerve-elements which are genetically independent of the former. (3) Nerve-cells in both vertebrates and invertebrates are connected with one another by broad anastomoses, constituting a network. (4) The neurone represents neither a trophic nor a functional unit."

Lugaro,[1] writing upon the present state of the neurone theory, concludes as follows: (1) The neurone can be disputed as a cellular unity, but it is surely an anatomic unit. The demonstration of neurofibrillæ and of their passage from one neurone to another does not destroy the conception of the anatomic individuality of a neurone. (2) This continuity has not been demonstrated in vertebrates; it is probable that such continuity among invertebrates is of a special sort, a kind of adaptation to a method of functioning not found among vertebrates. (3) The law of dynamic polarity which assigns to the cellular bodies and the dendrites a receiving function, and to the axones a discharging function, in its general outlines, is still intact; in some particular cases this law may be modified by the passage of neurofibrils or by the formation of the fibrillary network. (4) The Wallerian law, even though the multicellular origin of the axone and the autogenous regenera-

(1) Archiv. de Anatomia e de Embrol., III, pp. 412-437.

flexor muscles of the deep layer; sometimes pressure must be combined with lateral movements of the superficial muscles. If the reflex is present, extension of the great toe, or of all the toes, will be noticed. There is evidently no other muscle except the flexors that could be brought into display. The tibialis posticus, the peronei, have entirely different function than flexion or extension of toes.

"Since excitation of the flexors produces extension of the toes, the reflex is truly paradoxic. In thirty patients it was present together with Babinski's toe-sign. In ten cases Babinski's sign was present, and the paradoxic reflex was absent, or vice versa; and, as in nine cases Babinski's sign was distinct but the paradoxic reflex slight on the same side, in nineteen cases, as against twelve, the two reflexes showed a tendency to replace each other. In twelve cases of hemiplegia the paradoxic reflex was present mostly on the unaffected side." His reflex was first described at the October, 1904, meeting of the Philadelphia Medical Society[1] and there demonstrated. Examination of 200 normal individuals failed to show its presence. At this meeting its resemblance to Oppenheim's reflex was brought out in the discussion, in that Oppenheim uses the blunt end of a percussion-hammer and presses deeply into the muscle.[2]

Quinquaud's Phenomenon. Fürbringer[3] discusses the value of Quinquaud's sign with reference to its relation to the misuse of alcohol. The tips of the outspread fingers of the patient are placed perpendicularly against the palm of the examiner. After a few seconds slight tremors, essentially a phalangeal crepitation, may be felt, independent of anatomic joint changes. Individuals not showing the sign are not alcoholics. If the sign is only slightly present, a conclusion of alcoholic abuse is not justified.

Levicnik[4] investigated the Quinquaud phenomenon in 200 patients. He gets the sign by having the patient place his outspread fingertips against the fingertips of the examiner, the hands of the two observers being at right angles

(1) Journal of Nervous and Mental Diseases, 1905, p. 123.
(2) Gordon also reported his method of obtaining the new reflex in the *Revue Neurologique* (Nov. 15, 1904, p. 1083), this report being the same as that appearing later in *American Medicine*.
(3) Deutsche med. Wochenschrift, 1904, p. 27.
(4) Wiener klin. Wochenschrift, 1904, p. 51.

to one another. His conclusions are as follows: (1) The symptom appears in the majority of abstainers and in very moderate drinkers more or less clearly. The mere presence of the phenomenon does not necessarily indicate alcoholism. (2) Individuals who do not show the Quinquaud sign are with great probability not alcoholics. (3) Among twenty-two cases with a pronounced Quinquaud sign there was not one very heavy drinker. (4) There is no relation between tobacco and the phenomenon of phalangeal crepitation. (5) The connection between tremor of the hands and Quinquaud's sign is very striking. If the Quinquaud sign is negative, tremor is also absent. In no case in which the sign is present was tremor of the hands absent. The degree of Quinquaud's sign corresponds closely with the degree of tremor.

Hoffman and Marx[1] examined 1,018 individuals, using both Fürbringer's and Levicnik's method. The sign was absent in 52 per cent of the abstainers and moderate drinkers, in 42 per cent of drinkers and in 31 per cent of hard drinkers. It was sharply present in 10.8 per cent of moderate drinkers and 19 per cent of habitual drinkers. The absence of the sign, or its presence in moderate degree, are without meaning. Its presence in marked degree carries with it a probability of 3 to 1 that the patient is a strong drinker. The sign was absent in two cases of delirium tremens. In 137 cases with a marked Quinquaud sign, tremor of the hands was present only fifty-five times, and tremor was present 146 times without the slightest trace of a Quinquaud sign.

According to Max Herz[2] the crepitation is made in the sheaths of the flexor tendons. Such crepitation will take place in every one in greater or less degree. In alcoholics the motor unrest emphasizes this crepitation. This explanation is not like that given by Furbringer, who places the site of the tremor in the joints of the fingers.

Tremor is a better sign of alcoholism than Quinquaud's, according to Fürbringer,[3] who examined 500 cases. He found tremor of the hands as follows:

(1) Berliner klin. Wochenschrift, 1905, p. 19.
(2) Münch. med. Wochenschrift, 1905, p. 22.
(3) Berliner klin. Wochenschrift, 1905, p. 21.

	Abstainers.		*Drinkers.*	
Degree of tremor.	No. of cases.	Per cent.	No. of cases.	Per cent.
None or slight.....	194	91.5	18	8.5
Moderate	153	73	56	27
Intense	28	35.5	51	64.5

From this it follows that tremor may be lacking in outspoken drinkers. A moderate degree of tremor is without meaning, though it indicates the use of alcohol 3 to 1. On the whole, tremor of the hands is more characteristic and constant as a sign of the abuse of alcohol than Quinquaud's sign.

Blood-Pressure in Nervous and Mental Diseases. Haskovec had previously investigated a few nervous and mental diseases from the point of view of blood-pressure, but his articles for 1905[1] are based upon a very large number of observations. A detailed history of previous investigations by others precedes an account of Haskovec's own work, which was done with Gaertner's tonometer. The right hand of the patient was stretched out comfortably on a table at such a height that the forearm was on a level with the heart. In many instances the pressure was estimated in both arms. All measurements were taken at the same room temperature and only those cases were selected where there had been no loss of blood or disturbance of heart action and no thoracic disease whatever. Even the breathing was noted and regulated in so far as it was possible to do so. In drinkers and heavy smokers the average pressure was found to be 120-130 mm. of mercury. Marked exercise and psychic disturbance raises the tension 10 to 20 mm. A pressure higher than 120-130 mm. is rare. At night the pressure on an average is 5 or 10-15 mm. higher than in the morning. In women from 17 to 35 years of age the average pressure normally was 90 mm., being unchanged even in pregnancy.

The pressure in nervous diseases was estimated in 105 men and 49 women, the cases being reported in detail in tabular form, with tabulated summaries. In neurasthenia the pressure was found subnormal in asthenic forms and supranormal in affective forms. The blood-pressure has no causal relation to the disease. The symptoms of vertigo, feeling of cerebral pressure, headache and insomnia are

(1) Wiener med. Wochenschrift, 1905, p. 522, 568, 622, 668, 732, 783, 840.

not due alone to increased blood-pressure, but occur when the pressure is subnormal. Disturbances of sleep were found to occur indifferently whether the pressure was above or below the normal. Hence, pressure of the blood has nothing to do with sleep, except when it departs in either direction from the normal. In cases of traumatic neurosis the blood-pressure has no diagnostic value. In syphilis the pressure was found uniformly raised. Curiously enough, in arteriosclerosis with neurasthenia the pressure is only slightly increased. In epilepsy and general paralysis of the insane the pressure is lowered. At the present time, however, no laws can be deduced concerning blood-pressure, and no therapeutic suggestions made. Many more individual estimations of blood-pressure must be made in all sorts of cases.

Astereognosis. A case of functional astereognosis is reported by G. Rennie[1] in a stenographer 29 years of age, who felt one evening, while she was at work, her whole left side become cold and stiff, her left hand becoming particularly clumsy. This attack, which was preceded by no premonitory symptom whatever, and which was not accompanied by loss of consciousness, was followed by a loss of the stereognostic sense. This persisted eight or ten months and then disappeared. It was attributed by the author to a disturbance of functional activity in the right superior parietal convolution.

Osseous Sensibility. Attention was called to this sign under the name of pallesthesia in the Practical Medicine Series, September, 1904. Numerous articles concerning it have appeared during the year just passed. R. T. Williamson[2] concludes an account of his investigations as follows: "(1) In the normal condition the vibrating sensation, tested by a 6-inch tuning-fork, is probably always present at the three points usually tested: the styloid process of the ulna, the internal malleolus, and the inner surface and anterior edge of the middle third of the tibia. (2) In early tabes it may be lost in the legs before impairment of other forms of sensation occurs, and before ataxia or Romberg's sign can be detected. (3) In certain cases of spastic paraplegia it may be lost in the legs when other

(1) Review of Neurology and Psychiatry, August, 1904.
(2) Lancet, April 1, 1905.

forms of sensation are not affected. (4) In more cases of diabetes mellitus and chronic glycosuria it is lost when sensation in other respects is normal. (5) From these facts it is evident that sensation cannot be declared to be normal until the vibrating sensation has been tested. (6) Though the loss of the vibrating sensation is a symptom of considerable interest, it is at present too early to say what its diagnostic value will be in the future."

Osteoakousia (hearing by means of bony conduction of sound-waves from tuning-fork to the internal ear) is ambitiously and exhaustively discussed by W. Neutra,[1] who concludes that it is wholly independent of the so-called vibrating sensation or osseous sensibility. Osteoakousia demands the conduction of sound-waves by the bones to the ear, while osseous sensibility demands merely the perception of these vibrations in the bone itself by the medium of nerves of common sensation. Osseous sensibility is to be looked upon only as a modification of the pressure-sense. Tuning-forks ranging from 100 to 200 vibrations per second are the most suitable for testing bony sensibility.

Egger[2] reports a case of tabes in whom the hearing was well preserved in both ears, but in whom skeletal audition or osteoakousia was much reduced. According to Egger this case indicates that skeletal audition and vibratory sensibility are intimately related. A similar case was reported by him[3] in May, 1905. [The Editor agrees with the conclusions of Neutra that osteoakousia and osseous sensibility are entirely independent of each other.]

Marinesco[4] discusses vibratory sensibility from the clinical point of view. A tabetic in whom tactile sensation for the most part was normal was defective in his perception of vibrations over most of his bones. In another tabetic the sense was better maintained, but was lost over an arthropathy. It was lacking in a patient whose Rolandic area had been removed on account of epilepsy. The transmission of the sensation cerebralward is by nerves and not by bones. In the cord the track follows the posterior columns close to the gray matter. Hence, it is very frequent-

(1) Deutsche Archiv f. Nervenheilkunde, XXVIII, pp. 107-175.
(2) Revue Neurologique, 1905, p. 133.
(3) Revue Neurologique, 1905, p. 360.
(4) Bull. de la Soc. des Sciences Med. de Bucarest, 1904.

ly found associated with thermal and algesic disturbances and losses.

In a second article[1] Marinesco states that in affections of the posterior columns of radicular origin, notably in tabes, vibratory anesthesia is discovered in examination of the bones of the foot, leg or thigh. The bones of the pelvis, the vertebræ and sacrum, the thorax and the upper extremities are involved in only advanced stages of the disease. When vibratory anesthesia involves the bones of the lower or upper extremities, ataxia will be found present in these limbs, although it is certain that the ataxia is not caused by the vibratory anesthesia. In tabetics with arthropathy vibratory anesthesia is complete at the site of the lesion. When arthropathy exists in the preataxic stage vibratory anesthesia is less intense, and is found only over the diseased bone. In general, disturbances of superficial sensibility precede losses of vibratory sensibility, but there are exceptions to this rule. Indeed, the only sign in a given case of lesion of the posterior columns may be this vibratory anesthesia. In mixed tabes, accompanied with ataxia, vibratory anesthesia is the rule, and may settle a debated diagnosis. In mixed tabes of spastic type, vibratory anesthesia may constitute the sole symptom indicative of a lesion of the posterior columns.

Compression of the cord frequently causes disturbances of vibratory sensibility, especially if the compression is extensive. In such instances vibratory sensibility may be lost in all the bones of the lower extremities and of the pelvis. In compression of the dorsal cord, the vertebræ share the vibratory anesthesia. In some forms of paralysis coexistence of vibratory anesthesia and thermanesthesia is met with, while tactile sensibility is preserved or but very slightly affected. In organic hemiplegia, accompanied by disturbances of the superficial or deep sensibility, disturbances of vibratory sensibility are also met, especially in the upper limb. In anesthetic leprosy with trophic disturbances vibratory anesthesia is found in all four limbs. Sometimes it is more marked at the elbow than at the lower end of the radius. Conduction of the vibratory impulses is upward in the posterior columns, close to the gray matter. The skin, muscles and ligaments take no part in the transmission of osseous vibrations. The frequent dissociation of

(1) Soc. de Biologie, Comptes Rendus, 1904, p. 333.

cutaneous and osseous sensibility shows that each has its own peculiar nerves for the transmission of impulses.

Cytodiagnosis. Brissaud,[1] Grenet and Rathery advocate local anesthesia as a preliminary step in lumbar puncture. In itself it is so painful that many patients will not submit to it a second time. Moreover, the movement of the patient at the critical moment, often leading to a small hemorrhage, may by the admixture of blood ruin a cytologic examination. Ethyl chlorid is unsatisfactory, for its effects do not extend deeply inward. Instead it is best to inject very deeply 1 c. c. of a 1 per cent solution of cocain or of stovain. If cocain is used, it is well to wait four or five minutes before inserting the needle. If stovain is used, the needle may be inserted at once. Anesthesia is accomplished in three steps: (1) Half a cubic cm. by a Pravaz syringe is used to anesthetize the skin and subcutaneous tissue. (2) The needle to be used in lumbar puncture is next made use of, being inserted for one or two minutes into the deeper parts, and the remaining half cm. injected into the tissue. (3) The needle is allowed to remain in place three minutes or so after having been emptied, and is then thrust into the spinal dura.

Chauffard and Boidin[2] in an article entitled "A Year of Lumbar Puncture" recount their experiences based upon 140 cases and 223 punctures. Positive information was secured in seventy-nine cases. Five c.c. of cerebrospinal fluid were invariably withdrawn, and after centrifuging for fifteen minutes were examined cytologically. The results follow: In eleven tabetics nine showed lymphocytosis, and of nine cases of general paralysis all showed lymphocytosis. Thirteen cases of tuberculous meningitis without exception showed lymphocytosis, and in nine of them the tubercle bacilli were also found. In eight out of nine cases of herpes zoster an extraordinary degree of lymphocytosis was present. Three cases of epilepsy and two of cerebral tumor and carbonic monoxid poisoning were negative. One case of multiple sclerosis, negative at first, was positive some months later. In five cases of focal softening only one showed a lymphocytosis. Special weight is laid upon the differential diagnosis of blood-stained fluid. Cytodiagno-

(1) Revue Neurologique, 1904, p. 799.
(2) Gazette des Hôpitaux, 1904, p. 725.

sis is also of value in differentiating meningitis from acute infectious diseases with meningeal symptoms. The importance of repeated examinations in the same individual is emphasized. The only bad effects observed were vomiting (three times) and headache. The authors recommend median instead of lateral puncture.

Medner and Mamlock[1] report the result of their investigations in Von Leyden's clinic. According to them a lymphocytosis is present in progressive paralysis, tabes, cerebrospinal syphilis, cases with Argyll-Robertson pupils, cases with headaches in those showing a history of syphilitic infection, herpes zoster, sciatica, and parotitis. No lymphocytosis was found in poliomyelitis, syringomyelia, old hemiplegias, multiple neuritis, functional neuroses, compression myelitis, cerebral tumor or epilepsy. Those hemiplegics who have a luetic history have a lymphocytosis, whereas the others do not. Evidently syphilis is, then, the cause of the lymphocytosis. Of nine tabetics, six had a history of syphilis, and of these three showed a lymphocytosis and three did not. Of the other three, without a history of syphilis, two showed a lymphocytosis and one did not. Lymphocytosis was also found in two non-syphilitic cerebral tumors. The authors, therefore, conclude that it is dependent upon long-continued and strong irritation of the central nervous system and that the irritation may be an intoxication (as syphilis, uremia and tetanus), or some mechanical affair, as a cerebral tumor. Meningeal irritation, as such, does not seem to be the essential cause. On the whole, the authors are somewhat disappointed in cytodiagnosis. "The frequent presence of a lymphocytosis in syphilis, and the fact that by no means all cases of tabes show a lymphocytosis, makes it impossible in a doubtful case of tabes to depend upon cytodiagnosis to settle the question." In general paralysis lymphocytosis is more constantly present, but "this disease offers very little diagnostic difficulty. [The bias of Von Leyden and his school as to the non-luetic etiology of tabes in many cases, has tinged the judicial faculty of the authors somewhat in their estimate of the diagnostic value of the cerebrospinal fluid.—Ed.]

(1) Zeitsch. f. klin. Medizin., 1904, Heft. 1 and 2.

H. Rosenthal[1] proposes a new method for examining the *cells* of the cerebrospinal fluid, objecting that centrifuging distorts and ruins cells and spoils their staining qualities. Instead he uses a Zeiss counting slide, with a chamber 2-10 of a mm. deep and 4 mm. square, first diluting the fluid in a *melangeur* 1 to 10. For a counting fluid he uses methyl violet 0.10 grams, distilled water 50.0, and glacial acetic acid 2.0 gr. He found that in fifteen syphilitic and metasyphilitic cases there were an average of sixty cells to the cubic millimeter; whereas in fifteen normal or functional cases there were only 0.5 to 2 cells per cubic millimeter.

Skoczynski[2] examined the *pressure* of the cerebrospinal fluid in thirteen cases and obtained the same results as have been hitherto obtained by all observers. He was fortunately able during an epileptic seizure to measure the pressure and found it exceedingly high at the beginning of the convulsion. The pressure varied during the clonic spasms, and after the fit was over it fell to a very low point. He found, as Nissl, that its percentage of albumin was increased in general paralysis, and in all of these cases there was a lymphocytosis. He found cholin, a derivative of lecithin, in thirty-two out of thirty-five cases of general paralysis, in three cases of cerebral syphilis, in two cases of multiple sclerosis (a third case being negative), and in one case of epilepsy.

Cestan and Ravaut[3] report a case of pachy-meningo-myelitis of the conus terminalis in which the cerebrospinal fluid showed xanthochromia and coagulation *en masse,* with the presence of cellular elements. Only six cases of this sort are on record. Two factors are thought to figure in the explanation: (1) A meningeal inflammation, and (2) a hemorrhagic meningitis. Hence, the yellow color and the huge amount of albumin present, the latter resulting from the dissolution of red blood corpuscles. The two factors may coexist, constituting Babinski's fibrinous hemorrhagic meningitis, or during a meningo-myelitis in activity a hemorrhage may take place.

Mongour[4] finds that in icterus, even when very pro-

(1) Neurolog. centralblatt, 1904, p. 966.
(2) Neurolog. centralblatt, 1905, p. 40.
(3) Gazette des Hôpitaux, 1904, p. 985.
(4) Comp. Rend. de la Soc. de Biol. de Paris, 1904, p. 397.

found, no pigmentation of the cerebrospinal fluid results. Ducrot and Gautrelet[1] produced intense icterus experimentally in a dog and found absolutely no trace of bile-pigment after a week in the cerebrospinal fluid, which was as clear and limpid as spring water. If, however, the choroid plexus is functionally disabled by an injection of 3 cubic centimeters of a saturated solution of methyl violet in the internal carotid artery of the dog after temporary ligature of the external carotid, the cerebrospinal fluid becomes bile-stained. In twenty-four hours it is clear again.

Roussy[2] reports a case of subdural meningeal hemorrhage without modification of the color of the cerebrospinal fluid. Evidently, then, while blood in the cerebrospinal fluid indicates hemorrhage, its absence does not indicate that there has been no hemorrhage.

THE NEUROSES.

HYSTERIA.

Physiologic Theory of Hysteria. Sollier[3] finds the psychologic theory of Janet unsatisfactory. He believes it to be a disease of the cortex of the brain, the manifestations of which in turn are due to arrest of function of individual islands of cortex, or to association-paths between psychic and sensorimotor centers. If a motor area can no longer be called into action, paralysis of motion results, and if a sensory area can no longer be excited there will be anesthesia. Suggestibility is looked upon as due to a lack of counteraction between different centers. In hysteria the cortex seems partially asleep or benumbed, and the appearance of hysterial patients, their somnolence and somnambulism are thought to support this theory. The cause of the cortical inhibition is puzzling, but Sollier leans to the idea that it is some sort of intoxication.

Bernheim,[4] after detailing his conception of the disease, draws the following conclusions: (1) The hysterical at-

(1) Comp. Rend, de la Soc. de Biol. de Paris, 1905, p. 160.
(2) Revue Neurologique, 1905, p. 651.
(3) Journal de Neurologie, 1904, p. 1.
(4) Revue Med. de l'Est, 1904; Nos. 2 to 7 inclusive.

diagnosis at first was strongly in favor of hysteria. Thoma tacks constitute a psycho-physiologic exaggerated reaction, of emotional origin. (2) They may be primary, developing in a healthy subject following some special emotion, such as fright, anger, mortification, pain, variable in each individual. (3) They may be secondary, developing in the course of a disease, such as neurasthenia, psychosis, neurosis, organic disease, toxic or infectious diseases, always through the mechanism of an emotional cause created by the disease. (4) These attacks develop in subjects who react in a special and exaggerated manner to certain emotions— that is to say in subjects who have an underlying hysterogenic basis. (5) The sensitive-sensorial stigmata, described by writers as characteristic of hysteria (anesthesia, narrowing of the field of vision, ovarian neuralgia), and motor disturbances (paralyses and contractures), are not constant in hysterical subjects and are often met with in impressionable but not hysterical subjects. (6) The phenomena described under the name of visceral hysteria, cough, aphonia, hiccough, nervous vomiting, hemorrhages, polyuria, anuria, etc., are frequently met without hysterical paroxysms in non-hysterical subjects; these are the visceral psycho-neuroses. (7) There is no hysterical fever, or fever due to nerve action alone, pure and simple. (8) Many organic diseases of the nervous system, hemiplegia, paraplegia, with exaggerated reflexes or with amyotrophy, peripheral neuritis, with association of dynamic nervous phenomena, have been wrongly considered by authors as pure cases of hysteria simulating organic diseases. . . . (10) Hysteria is not a morbid entity. This word should not be deflected from its primary meaning to be applied to innumerable psychoneuroses of emotional origin, suggestive or traumatic; it should be reserved for the crises which certain subjects pass through on account of certain emotional causes and which are often apt to be caused also by suggestion or auto-suggestion.

Hysteria and Organic Disease. Thoma[1] reports in detail four cases of nervous disease: tuberculous meningitis, multiple foci of cerebral softening, multiple sclerosis, and carcinomatous cerebral metastases. In each of these cases the diagnosis at first was strongly in favor of hysteria. Thoma

(1) Zeitsch. f. Psych. LX, p. 606.

insists, too, that in all such cases there is a true hysteria present which develops with the organic lesion. The claim of Kraepelin that hysteria is invariably congenital is not in accordance with the facts. For example, trauma and poisons may cause true hysteria, though it is admitted that they may the more easily do so, the greater the predisposition. Thoma believes that metabolic disturbances play a part in causing hysteria. In simple psychoses, accompanying hysterical symptoms are to be looked upon as the expression of a specific disturbance of nutrition.

Levi and Taguet[1] report a case showing cerebellar symptoms and evidence of organic nervous disease mingled with other symptoms of an hysterical nature. According to Babinski in hysteria only those symptoms occur which the will is capable of reproducing. Since in this case the patient showed the sign of combined flexion of thigh and pelvis, and the so-called oculo-pupillary syndrome (narrowing of the palpebral fissure, enophthalmus, and slight myosis), organic disease is present. The oculo-pupillary syndrome would indicate a lesion in the medulla.

Hysterical Symptoms. (For behavior of the *plantar reflex* see Crocq's article, p. 1020.

Hysterical Fever. Dircksen[2] is willing to admit the possibility of hysterical fever, but in most cases it is simulated by various artifices. In the latter group of cases other symptoms are also simulated, such as dyspnea, syncope, hemoptysis and vomiting. In these individuals a morbid mental state exists. Many have as a fixed idea that of being in a hospital. Bernheim[3] does not believe that there is such a thing as hysterical fever. The strongest emotions and the greatest nervous shocks do not in themselves determine fever.

Anuria. Cestan and Nogues[4] report a case showing anuria, and even the signs of uremia, without any evidence of any organic cause whatever for the phenomenon other than hysteria. The case occurred in a girl of 19 years, who had previously had several hysterical accidents. Important is the fact that the anuria suddenly disappeared by suggestion. The pathogenesis of such hysterical anuria is left undecided by the authors. Spasm of the glomerular

(1) Revue Neurologique, 1904, p. 1,237.
(2) Revue Neurologique, 1905, p. 194.
(3) Revue Medicale de l'Est., 1904, 1.
(4) Revue Neurologique, 1905, p. 358,

vessels, inhibition of the bulbo-spinal centers, general disturbances of metabolism, are suggested as possibilities.

Aphasia, Aphonia and Mutism. Courmont[1] reports a case of hysterical aphasia, calling attention to Charcot's statement that it does not exist, all cases being those of hysterical mutism. Hysterical aphasia as such was first insisted upon by Raymond and Janet in 1898 in their book, "Neuroses and Fixed Ideas," though other cases had been reported by Ballet, Lépine and Sollier. Courmont's case occurred in a girl of 22 years, an alcoholic, who showed aphasia with aphemia, agraphia, word-deafness and word-blindness, without psychic blindness. She also showed disturbances of memory, right-sided hemiplegia and hemianesthesia. Noteworthy was the dissociation of aphasia for French and for German, both of which she spoke. The author's conclusions follow: "This case proves the existence of hysterical aphasia, with all the qualities of organic aphasia. (2) Hysterical mutism, as described by Charcot, is merely an aphasia of transmission (aphemia), being complicated with agraphia, word-deafness or word-blindness. Hysterical mutism, far from being a distinct entity, belongs in the larger class of hysterical aphasia. (3) There is, in hysterical subjects, a form of mutism which is due to the will of the patient. For this voluntary mutism the term 'hysterical mutism' should be reserved. In hysterical aphasia the patient cannot speak; in hysterical mutism he *will* not speak."

Stinzing[2] regards hysterical mutism as very rare and reports a case, combined with hysterical asthma, occurring in a young man of 29 years after an accident. In aphasia of cerebral origin, and even in hysterical aphasia, as a rule single words or syllables at least can be uttered, but phonation is completely gone in hysterical mutism. The intelligence is unaffected. This patient showed no agraphia and communicated his thoughts in writing. Sometimes hysterical stammering is associated with hysterical mutism, but not in this case.

Hysterical Sleeping States. Grober[3] reports the case of a child of 3½ years, which after numerous attacks in all

(1) Lyon Med., June 18, 1905.
(2) Deutsche Archiv f. Nervenheilkunde, XXVIII, p. 273.
(3) Deutsche Archiv f. Nervenheilkunde, XXVIII, p. 293.

probability of a hysterical nature, fell in a lethargic condition, lasting for three months, associated with lively choreic movements and loss of consciousness, possibly ushered in by an organic disease (meningitis). The child suddenly awakened spontaneously, but had lost its powers of speech and suffered from a purely functional stiffness of the right wrist, all clearing up in a short time. A few other hysterical seizures of short duration followed. The child has now been healthy for a year and a half.

Raecke[1] reviews the subject of the hysterical sleeping state, based upon his experience with twenty-three cases. It is often preceded by mental dulness or headaches. Often there are paresthetic sensations in the hands and feet, extending gradually to the head. In sleep the face is expressionless, the gaze fixed, but frequently the eyes are closed. Sometimes the limbs are flaccid, sometimes there is *flexibilitas cerea*. The tendon reflexes are preserved, and are often increased, whereas the cutanous reflexes are reduced or absent. The temperature is normal. The color is normal or heightened, seldom pale. The pulse is full and equal; the respiration is often superficial. Cyanosis, however, is exceptional. The pupils are dilated and equal, but may be of medium diameter, and the pupillary reactions are normal. The tactile and pain-senses are abolished, but there may be hyperalgesia over the skull and the bottoms of the feet. Consciousness is not wholly lost, nor is there a simple, dream-filled sleep. There is rather a very limited state of consciousness. The patient seldom soils himself, but by his restlessness indicates his needs. On the other hand, he will not eat and has to be fed often through the nose. The awakening is usually sudden, but is often gradual, consciousness being restored by degrees, and there is often preserved recollection of the events which happened during the sleeping state. The duration is hours, days, weeks, rarely months. A bibliography follows the article.

Vasomotor Disturbances in Hysteria. Genevrier[2] reports the case of a girl of 23 years, showing all of the usual hysterical stigmata, and in addition presenting upon her abdomen, buttocks, backs of her hands, fingers and

(1) Berliner klin. Wochenschrift, 1904, p. 1323.
(2) Nouvelle Iconographie de la Salpêtrière, XVII, p. 459.

left calf large patches of superficial gangrene, followed by whitish keloid-like scars. The author watched the development of the patch on the left calf. There was first an intense vaso-constriction. The limb was cold and the toes resembled in appearance the *doigt mort.* The skin was mottled, cyanotic in places and looked and felt like the skin of a cadaver. Auto-mutilation in this case is harder to believe than spontaneous superficial gangrene. The surface of the gangrenous patch was uniform, and so was the depth. A caustic could hardly have acted so uniformly. The author explains the ulcerations as due to prolonged vascular spasm.

Hysterical Intestinal Occlusion. Sakarraphos[1] reports a case, recurring several times, associated with meteorism, atrocious pain and fecaloid vomiting, in a girl of 17 years. The patient had never before passed through any hysterical crises nor peculiar mental condition. There was present, upon examination, narrowing of the field of vision, a focal reduction in sensibility and slight ovarian pain on pressure. A suggestive treatment resulted successfully.

Weber[2] reports a similar case of fecal vomiting and reversed peristalsis in a girl of 22 years. She gave a history of vomiting (sometimes with a little blood), distension of the abdomen, and great constipation. At the time of the attack "actual scybala or formed feces from the large intestine were certainly vomited on more than one occasion. . . . At times, when an oil enema was administered, some of the oil reappeared in the vomited matter. In order to guard against and to obtain an accurate diagnosis an enema colored with methylene blue was administered by the nurse. Some of the methylene blue appeared in the vomited matter within ten minutes after the administration of the enema." Notwithstanding the diagnosis, two exploratory laparotomies were done, and at neither of them was any abnormal condition whatever detected. In the latter operation the stomach itself was opened and explored. Subsequently the patient came under the care of Sir Frederick Treves and he did a third laparotomy without any positive findings, his object being chiefly to explore the colon.

(1) Progrès méd., 1904, p. 497.
(2) Brain, 1904, Summer, p. 170.

Other cases are reviewed from the literature and the following conclusions drawn: (1) Functional nervous vomiting, like the hemianesthesia, palsies and spasms of hysteria, must be regarded as due to an abnormal state of the cerebral cortex, and is just as much a symptom of functional brain disease as the vomiting in cases of cerebral tumor is of organic brain disease. (2) Fecal vomiting of functional nervous origin is merely a rare and extremely exaggerated form of ordinary hysterical vomiting. (3) The vomiting in functional brain disease may sometimes be more violent and severe than it ever is in organic cerebral disease, since fecal vomiting is scarcely known to occur in cases of cerebral tumor, etc. (4) For the occurrence of fecal vomiting of functional nervous origin active internal antiperistalsis is absolutely necessary. But it is not certain that antiperistalsis necessarily always plays a part in the fecal vomiting known to surgeons as a symptom of organic intestinal obstruction (organic ileus). (5) The fecal vomit in organic obstruction of the bowel is seldom, if ever, more than "feculent"—that is to say, sharing the odor of feces without containing obvious (visible) fecal particles or masses. Vomiting of formed feces in the absence of malingering and gastro-colic fistula practically occurs only in functional nervous cases. This may partly be accounted for by remembering that antiperistalsis, if it occurs at all, is likely to be more forcible when the muscular walls of the gut have not been previously weakened by overdistension or gross organic disease. (6) "Hysterical malingering" is, of course, apt to develop in the same (hysterical) class of patients in whom fecal vomiting occurs, and the possibility of genuine fecal vomiting occurring side by side with simulation must be kept in mind.

Akinesia Algera. L. Ingelrans[1] has written an extensive essay upon akinesia algera, which term fairly well carries its meaning within it, viz.: inability to move without pain. In patients showing this syndrome first described by Moebius, no cause can be found which reasonably accounts for the pain. All of the cases in the literature, beginning with the two original cases of Moebius, are reviewed. It is looked upon more as a syndrome than as a morbid entity.

(1) Gazette des Hôpitaux, 1905, p. 783.

Neurasthenia. Leubuscher and Bibrowicz[1] have made a study of neurasthenia in the working classes, and find that there is an enormous and ever-growing increase in the number of cases of neurasthenia in the working classes of great cities. Three-fourths of such cases are acquired cases. The maximum incidence of the disease is between 25 and 45 years, which is the time of the greatest earning capacity. The more intellectual the occupation, the greater the number of neurasthenics. A whole series of difficult sociologic questions underlie the disease, and they must be settled before there can be any prophylaxis. Sanatoriums for the nervous should be provided at the public expense. [These remarks, of course, apply to Germany. —ED.]

Hunt[2] believes that neurasthenia can best be *cured* by exercise and forced feeding, illustrating his remarks by two cases. "The patient's day began at 7, with a cold plunge and a glass of milk. At 8 a. m. he received breakfast, and then was immediately taken for a brisk walk of about 5 miles. At 11:30 a. m. he received a second glass of milk with an egg, then rested until 12:30, at which time he was interested mentally by cards or books until 1 p. m., when dinner was served. At 2 p. m. exercise by golf, tennis or croquet was prescribed. At 5 p. m. another glass of milk and egg were taken; at 6 p. m. more exercise was indulged in, followed by a cold shower bath, and at 7 p. m. supper was served. The evenings were devoted to games and conversation."

M. Page[3] reports five cases of neurasthenia in which epigastric pain is the dominant symptom. The pain is extremely violent, and is either three-finger breadths below the ensiform cartilage, or in the back at the level of the eighth dorsal vertebra.

A. Schott,[4] after thoroughly discussing the general subject of hypochondriasis and hypochondriac states, concludes as follows: (1) Hypochondria is not a nosologic unit, but is instead an expression of degeneracy. (2) Hypochondriac states are frequently found in hysteria and neurasthenia. (3) They occur occasionally in all forms

(1) Deutsche med. Wochenschrift, 1905, p. 820.
(2) Journal Amer. Med. Ass., June 3, 1905.
(3) Progrès Médical, 1905, p. 24.
(4) Berliner klin. Wochenschrift, Dec. 19, 1904.

of mental impairment. (4) In dementia precox the hypochondriac states, on account of the danger of confounding them with simple neurasthenia or hysteria, demand an especially careful clinical examination and analysis. (5) In severe hypochondriac states the danger of suicide or self-injury is never to be lost sight of. . . . (7) In all hypochondriac conditions a careful physical examination is doubly indicated, and all peripheral causes of irritation are to be removed as far as possible.

PSYCHASTHENIA.

Psychasthenic anorexia is described by Buvat,[1] and is to be distinguished from hysterical anorexia and the sitophobia (aversion to food) of the insane. Raymond reported a case in 1902, and gave as points differentiating it from hysterical anorexia the absence of epigastric, gastric or pharyngeal anesthesia. Since then Buvat has observed five cases, these forming the basis of his article. The psychasthenic sees his dinner hour arrive with feelings of anguish, his mental depression and his phobia-obsession become worse and worse each moment; he will struggle to keep food from being introduced into his stomach; he will clench his teeth; and sometimes it is necessary to have recourse to the stomach tube to overcome his obstinacy. He will vomit three or four times in succession the things which he is successively forced to take; he will not become accustomed to the tube, because his naso-pharyngeal sensibility, unlike the case in hysteria, persists, and the tube is more painful than normal mastication and deglutition. When he has had his forced meal he breaks out into complaints, he is going to stifle, a red-hot iron is gnawing into him. The anxious state lasts all through the digestive period. "Even after the patient has been forced to gain a dozen pounds the anxiety-phobia still persists, and the normal mental state is not regained till long after a decided improvement in the physical condition. In psychasthenia one cannot foresee in what manner the disease will terminate, and the prognosis has to be left in abeyance; there is a dissociation between the mental and

(1) Gazette des Hôpitaux, 1905, p. 639.

physical condition, the latter always improving with food, but the former more slowly, with setbacks."

Psycholeptic Crises. Janet[1] last year read before the Boston Society of Psychiatry and Neurology a paper devoted to a set of crises occurring in psychasthenics, of an epileptic nature, to which he and Raymond have given the name "psycholepsy." In psychasthenics showing these crises, the mental trouble does not develop slowly and insidiously, but rather stormily, reaching a climax sufficiently quickly to constitute a veritable crisis. A number of cases are given in illustration. Some of the crises are of short duration and may be followed by a more or less complete restoration of mental activity, as in epileptics; or they may be the point of departure for a long mental enfeeblement. Negatively there are three important factors: (1) There is no delirium properly so called, and not even any mental confusion; the patient expresses himself clearly and reasons intelligently. One even notices an increase in the memory for the past and in the subtlety of the reasoning faculty. (2) There is an absence of any actual motor trouble; the subject moves perfectly in every way. The tendon and cutaneous reflexes are normal throughout. (3) The different forms of sensation are perfectly intact, so far as we can at present determine.

The positive characteristics are two: (1) The peculiar feelings expressed by the patient in regard to all his cerebral operations, feelings which cause him to say that everything about him is incomplete or unfinished; that things have no reality, even his own person shrinking and becoming as nothing. They are possessed by "a feeling of incompletion." (2) The second important positive characteristic is the disturbance of action. The alteration of movement itself or of the sensation of movement is not the essential factor in these crises; it is rather the alteration of action. All sorts of disturbances of the will, resolution, and attention occur. If the crisis is somewhat prolonged, there may be observed every variety of abulia, irresolution, feebleness of effort, and disorder of action.

The absence of genuine motor disturbances, of all paralysis, of all alteration of the reflexes and of sensibility,

(1) Boston Medical and Surgical Journal, 1905, p. 93.

serves to rule out most of the graver cerebral lesions. The disturbance of attention involves only present or recent events and leaves intact the reasoning power and memory of the past, thus permitting elimination of states of mental confusion and stupor. The differentiation from the hysteric crisis is made easy by reason of the fact that after the psycholeptic attack the patient retains a very clear memory of what he has been through. In hysteric crises amnesia plays a much greater rôle, and there is no feeling of incompletion and no abulia. In epilepsy, much harder to differentiate, the mental disturbance in general is more profound, the intelligence disturbed to a greater extent and the consequent amnesia more marked.

Raymond and Guillain,[1] at a meeting of the Société de Neurologie de Paris, reported a case of stasobasophobia in a psychasthenic, and Raymond[2] again recounted the same case in the *Bulletin Medical,* under the caption, "A Case of Stasobasophobia in a Tuberculous Psychasthenic, Showing a Myopathy." The young man, 20 years of age, with a tuberculous ancestry on both sides, and suffering from a chronic tuberculous otitis, was kicked by a horse in the right thigh. Following that both legs became weak. When he stops walking or when he wishes to stand up, his legs flex, he gets into an anxious state, and falls unless held up. The weakness of the legs in this case is real, and his ataxia is not hysterical, but is a true phobia.

Mental Torticollis. Massary and Tessier[3] presented at a meeting of the Société de Neurologie de Paris a case of mental torticollis or spasmodic torticollis, in a nervous woman, with a torticollis obsession, in whom the reflexes were exaggerated. The anxiety shown by the patient, according to Brissaud, stamped the case as mental.

Pitres[4] reports a similar case treated unsuccessfully by several physicians and finally cured by a quack. He does not quite agree with Brissaud, who first described the condition under the name "mental torticollis," that the origin is wholly mental, but thinks that it has some analogy with functional spasms, such, for example, as writer's cramp.

(1) Revue Neurologique, 1904, p. 1231.
(2) Bulletin Medical, XIX, p. 45.
(3) Revue Neurologique, 1904, p. 1204.
(4) Journal de Méd. de Bordeaux, 1904, p. 665.

The quack in this case effected the cure by suggestion and vigorous and painful rubbing of the neck.

EPILEPSY.

The Relation of the Epileptic to the Community. Bullard[1] believes that the highest proof of civilization is the care shown for the weak, disabled, and unfit. Pauper epileptics, too, form a danger to the community. A certain number of them are apt to commit atrocious and causeless crimes. Many of them are shiftless, irresponsible, drunken and vagrant. They must be supported in their homes in idleness, and their mental condition, irritability, and unreasonableness render them undesirable inmates of the family. It is better for both the epileptic and the family that he be supported elsewhere.

Copp,[2] in reviewing the state's relation to the epileptic, believes that the epileptic has the same rights of protection and education by the government that the normal individual has. There can be no limitation of responsibility by the state dependent upon achievement; the weak and defective are also citizens. If the epileptic is denied the privilege of a public-school education, or of obtaining a training fitting him for self-support, and he therefore should fail in his pursuit of life, liberty and happiness, the state is at fault. Moreover, the activities of the state must extend to humane and charitable undertakings, and our conception of public duty being constantly enlarging, we may later look upon our charity of the present as mere justice in the future. The state should be alert to discover conditions adverse to the welfare of its citizens, and then to correct them.

Epilepsy is a menace from many points of view. Many epileptics show criminal propensities. Again, although epilepsy shows a strong tendency to self-extinction, with an excessive infant mortality, it is unfortunately kept alive by the regenerative forces inflowing through healthy persons marrying epileptics. Hence, numerous interchangeable forms with neuropathic taint, such as insanity, idiocy,

(1) Boston Medical and Surgical Journal, 1905, p. 123.
(2) Boston Medical and Surgical Journal, 1905, p. 124.

chorea, and hysteria, are perpetuated. The state's duty should provide: (1) a center for study, research and teaching, in relation to epilepsy; (2) colonies, independent or associated with such a center, which the adult epileptic may enter and find a home in place of social ostracism; (3) custodial provision for the intractable and insane epileptics. Marriage of epileptics to each other or to the healthy should be prohibited by all states. Thus far only Connecticut, Nebraska, Minnesota, and Pennsylvania have restrictions and punitive legislation. The law of Connecticut, enacted in 1895, follows:

"*Section 1.* No man and woman, either of whom is epileptic, imbecile or feeble-minded, shall intermarry or live together as husband and wife when the woman is under 45 years of age. Any person violating, or attempting to violate, any of the provisions of this section shall be imprisoned in the state prison not less than three years.

"*Section 2.* Any selectman or any other person who shall advise, aid, abet, cause or assist in procuring, or countenance any violation of Section 1 of this act, or the marriage of any pauper when the woman in such marriage is under 45 years of age, shall be fined not less than one thousand dollars or imprisoned not less than one year or both.

"*Section 3.* Every man who shall carnally know any female under the age of 45 years who is epileptic, imbecile, feeble-minded, or a pauper, shall be imprisoned in the state prison not less than three years. Every man, who is epileptic, who shall carnally know any female under the age of 45 years, and every female under the age of 45 years who shall consent to be carnally known by any man who is epileptic, imbecile or feeble-minded, shall be imprisoned in the state prison not less than three years."

Sir Wm. R. Gowers[1] reviews the *relation of epilepsy to life insurance.* Epilepsy is a rare cause of insurance claims, and it is rarely the direct cause of death to those who suffer from it. It may be an indirect cause by the accidents to which it leads. Life insurance as a rule seldom begins before 20 years, before which time three-fourths of the hereditary cases will have manifested themselves. It is, however, a question whether all epileptics are uninsura-

(1) Lancet, 1904, p. 1061.

ble. Gowers believes that some cases should be accepted on the payment of extra premiums. He does not think that a family history of epilepsy in a candidate need lead to extra premiums or a refusal to insure, provided the candidate himself be free from flaw. Previous attacks of epilepsy in a candidate are generally equivalent to the present existence of the disease. Moreover, there is in epilepsy a danger of suicide.

Cholin in Epilepsy. J. Donath,[1] who has previously written upon the subject, again reviews the significance of cholin in epilepsy, based very largely upon his own examination of the cerebrospinal fluid. In 18 cases of genuine epilepsy, cholin was found 15 times. In 3 cases of Jacksonian and one of syphilitic epilepsy, cholin was uniformly found. [His article, translated into English, was published in full in an American journal[2] and can easily be referred to by the reader, who wishes a thorough account of the chemistry of the subject.—ED.]

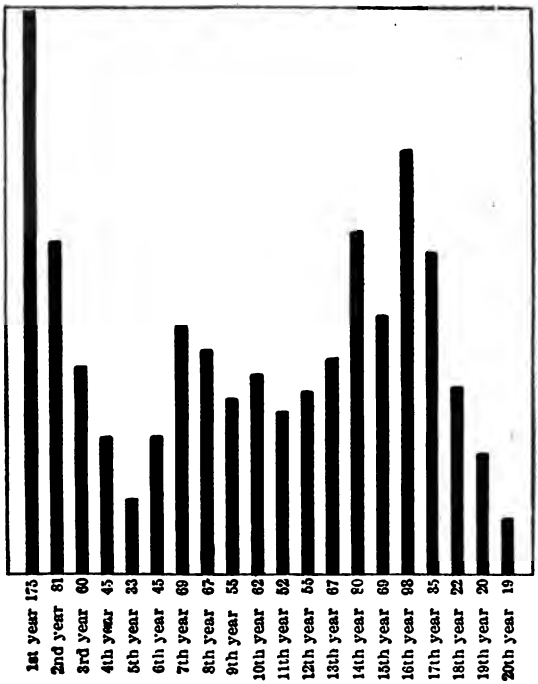
Etiology. The influence of the first and second dentition periods in the etiology of epilepsy is reviewed by Spratling.[3] He believes (very properly) that we are never justified in looking upon the convulsions of infancy and early life as harmless manifestations. The three charts which follow are instructive in this connection, based as they are upon the statistics of the Craig Colony of Epileptics.

Six cases are related in detail. Spratling summarizes his conclusions as follows:

"1. Difficult dentition, *i. e.*, the piercing of the gums by the tooth may, in suitable subjects, constitute a sufficient irritant to cause convulsions. 2. In suitable subjects these convulsions may ultimately lead to epilepsy. 3. By suitable subjects I mean infants who inherited a neuropathic tendency to disease; whose parents had epilepsy, or insanity, or who were alcoholic, or suffered from some other general vice that could be transmitted to the offspring in some other form capable of vitiating its powers of resistance to disease. 4. I do not believe that difficult dentition alone in a child who inherited no ancestral taints, and

(1) Deutsche Archiv f. Nervenheilgunde, XXVII, p. 71.
(2) Medical News, 1905, p. 107, 161.
(3) Medical News, 1904, p. 1111.

Chart 1.



Showing the age in years at which epilepsy originated in 1,215 cases. Note the rise during the six, seven and eight years, coincident with the second dentition period.

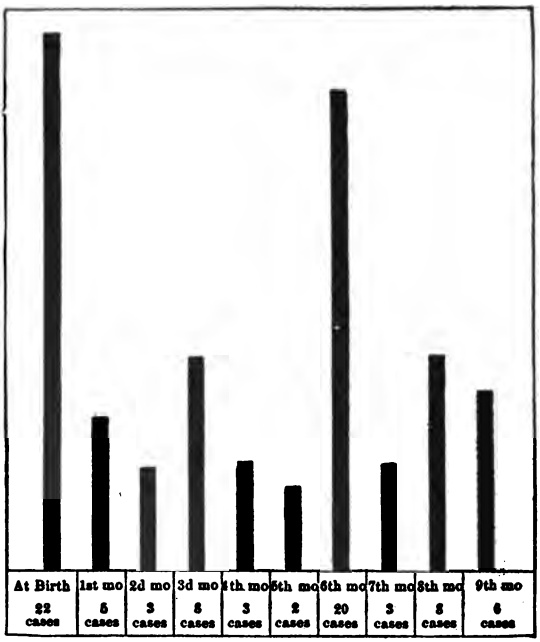
who at its birth is free from a tendency to nervous disease, can cause epilepsy. 5. Great caution must always be exercised to lay the true cause in cases of this kind where it belongs; for the reason that gastrointestinal disorders, the sequelæ of the eruptive fevers and other factors common at this age, may produce similar results."

The influence of puberty and adolescence in their relation to the etiology of epilepsy is discussed by the same author,[1] with the following conclusions:

"First—That we can, and must, in many cases of epilepsy that appear during the twelfth to the sixteenth and

(1) Spratling, N. Y. Med. Jour. & Phila. Med. Jour., 1905, p. 995.

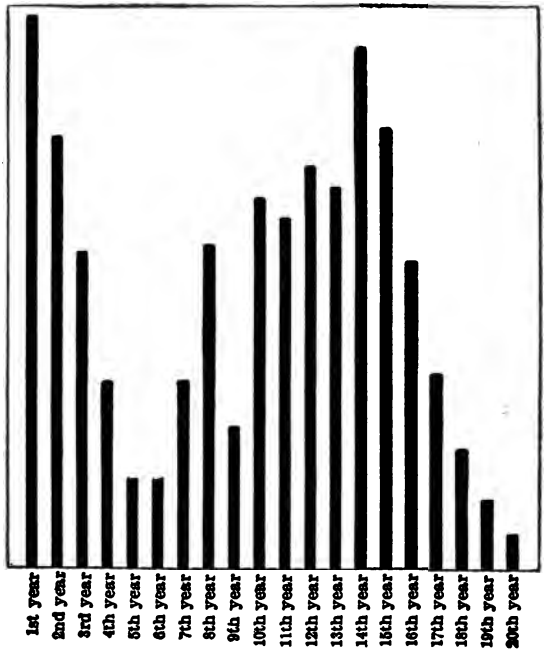
Chart 2.—First Dentition.



Showing the age in months at which epilepsy originated in 80 cases. Note the increase at the sixth month, which marks the beginning of the influence of the first dentition, that is, the period when the teeth begin to cut through the gums.

eighteenth years, coincident with the establishment of the menstrual flow in women, and with the passing of boyhood into manhood, ascribe to these changes the power of inducing well defined convulsions that may be epileptic. Second—That except in the most remote and exceptional instances, these periods alone in normal individuals have no power to induce epilepsy or even epileptoid phenomena. Third—That by searching carefully we shall find in most cases of epilepsy at this period either a previous history of convulsions, usually in infancy, or a family and personal history so tainted with a tendency to disease that epilepsy under the stress of puberty is plainly invited."

Chart 3.



Showing the age in years at which epilepsy developed in 3,523 cases. Note the greatest number during the first year, the increase during the seventh and eighth years, and the still greater increase during the epoch of puberty.

Leser[1] investigated the subject of *epilepsy* and *organic heart disease,* and found in 53,980 patients at the Policlinic in Prague 527 of the former and 814 of the latter. In only 8 cases, however, did epilepsy and cardiac disease coexist. He therefore concludes that cardiac epilepsy scarcely exists. The association of *epilepsy* and *migraine* is insisted upon by Epstein[2] and Turner.[3] The latter believes that some forms of migraine should be classified as epileptic equivalents. Noteworthy is the frequency of migraine in the ancestry of epileptics, the presence of peri-

(1) Neurolog. Centralblatt, 1905, p. 224.
(2) Ibid., 1905, p. 225.
(3) Lancet, 1905, p. 706.

odic headaches in epileptics, and the not uncommon substitution of migraine by epilepsy. *Trauma* as a cause of epilepsy is insisted upon by Brush,[1] who reports 38 cases.

Pathology. Turner[2] believes that the most striking changes are those recently described by Clarke and Proud in the sensory cells of the second cortical layer. Dr. John Turner of the Essex County Asylum has found coagula in the veins, capillaries and arterioles of the cerebrum and cerebellum, apparently made up of amalgamated blood-plates. These thrombi deprive the cortical cells of their necessary blood. The essential feature of the development of convulsions would appear to be deprivation of arterial blood, coinciding with capillary and venous stasis, from cortical motor areas. Concerning the study of the blood, Turner is quoted as follows: "Haig suggested that there was a definite relation between excess of uric acid in the blood and such maladies as migraine and epilepsy; but treatment directed upon this theory had not proved of greater value than other methods of treatment conducted along conventional lines. Somewhat similar to the uric acid theory is the view of Krainsky, who propounds the doctrine that uric acid is not the toxic agent, but a derivative in the form of ammonium carbamate, a substance having a convulsion-producing character, as testified by its injection into rabbits. Pugh has found a sudden fall in blood alkalinity immediately prior to a fit, and a further diminution in the alkalinity after the convulsion, the average interparoxysmal alkalinity being lower than the average of normal cases.

"The general conclusion to be derived from a study of the researches upon the blood in epileptics is that prior to a convulsive attack or a series of attacks there is a diminution in the blood alkalinity, but whether this arises from retention of uric acid (Haig), from formation of carbamic acid (Krainsky), or from the presence of acid toxins as yet of undetermined nature and source, remains unproven. The stage of diminished alkalinity would correspond to the preparoxysmal period of clinical observers—a period characterized in some cases by mental phenomena such as irritability, lethargy, depression, and occasionally

(1) Journal of Nervous and Mental Diseases, 1905, p. 245.
(2) Lancet, 1905, p. 706.

delusional states, and in other, though rarer, instances by an unusual sensation of well-being. It is not to be supposed that such symptoms are invariably present or are characteristic of all forms of epileptic seizure. They are found more especially in serial epilepsy—that is, epilepsy shown by frequently recurring fits for a few days, to be followed by an interval of freedom of greater or less duration; in cases in which two or three fits occur at a time; or in cases the type of which is an occasional severe convulsive attack. They are not present, so far as I am aware, in the *petit mal* type of the disease."

B. Onuf[1] reports some interesting autopsy findings in 16 epileptics from the pathologic laboratory of the Craig Colony. Of 15 of these cases 12 showed distinct changes in one valve or the other. Onuf looks upon the valvular changes as secondary to the epilepsy; being brought about by the strain to which the circulatory system is put in a *grand mal* seizure. Study of the capillaries showed striking tortuosity with aneurismal dilatation, doubtless due to the same strain. In 10 cases the pia was thickened over the hemispheres. In one case there was circumscribed atrophy of the right frontal lobe, in another subdural hemorrhage over the posterior part of the second and third frontal convolutions and lower part of the central convolutions; in another internal hydrocephalus; in another a cerebellar cyst; in three cases shrinkage of the cerebellar convolutions; in 7 of 9 cases distinct changes in one or both thalami. The changes in the thalamus and geniculate bodies were in the nature of an atrophy.

Orloff[2] gives a series of very complete examinations of the brain of epileptics, and describes 3 varieties of neurogliar sclerosis of the superficial layers of the cortex, the focal form, the generalized form, and the form described by Weber in which the superficial layer remains thin, but in which the whole external layer consists of slender and of broad fibers, arranged mostly irregularly, but to some extent radially, all being crowded too closely together to form a true network. The gliosis is specially marked in the horn of Ammon in the stratum granulosum.

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(1) Journal of the Am. Med. Assn., 1905, p. 1325.
(2) Archiv f. Psychiatrie, XXXVIII, Heft. 2.

Status Hemiepilepticus Idiopathicus. Leo Müller[1] reports 8 cases of this very interesting condition. Despite the post-mortem examinations made in the fatal cases, no anatomic explanation was found for the symptom.

Symptomatology. The so-called *"aura canora,"* an aura of singing, is reviewed by Bianchini,[2] who adds to the 4 cases thus far appearing in Italian literature 3 cases of his own, two in females and one in a male. In the first of the three cases the terminal phase of the attack, which is very long (eight to ten days), consists of a psychomotor excitation betraying itself in song. In a second case, just preceding the attack, the patient begins to walk furiously, his face grows red, the pulse rises above 100, and he begins to sing. The "aura canora" is essentially a motor phenomenon which passes over into a psychomotor and psychic phenomenon.

Newmark[3] reports a case showing a series of nearly 600 spasms in an epileptic without disturbances of consciousness in the intervals. The absence of any affection of consciousness or of the intellectual or emotional state after the body had been racked by hundreds of bilateral convulsions, is extraordinary.

Hultgen[4] discusses the *psychic equivalents* of epilepsy, and enumerates as the chief examples: (1) vertiginous attacks, a kind of mental disorientation; (2) absences, in which there is a temporary shutting off of consciousness; (3) attacks of amnesia, of sudden and temporary nature; (4) the dreamy state of Hughlings Jackson or the automatism of Gowers; and (5) the impulsive states, homicidal, suicidal, destructive, or states of pyromania, kleptomania, dromomania, dipsomania, etc.

Diagnosis. Putnam and Waterman[5] discuss the differential diagnosis between epilepsy and hysteria; citing 11 illustrative cases. Bratz and Falkenberg[6] are of the opinion that a positive diagnosis of epilepsy and hysteria can always be reached if sufficient care and pains are taken, but that it must sometimes remain in abeyance for long pe-

(1) Deutsche Archiv f. Nervenheilkunde, XXVIII, p. 31.
(2) Revue Neurologique, 1904, p. 970.
(3) Medical News, Nov. 29, 1904, p. 828.
(4) Illinois Med. Journal, December, 1904.
(5) Boston Medical and Surgical Journal, May 4, 1905.
(6) Archiv f. Psychiatrie, 1904, Heft. 2.

riods, even of several years' duration. There is no single mark, not even the pupillary reaction, which can be regarded as decisive. Each case must be studied in all its features, and between the seizures as well as in them. The 11 cases described by Putnam and Waterman are well worthy of careful examination.

Prognosis. Wherry[1] believes that individualization of treatment in epilepsy, with change of environment, will double the percentage of recoveries. W. A. Turner,[2] in an article published both in Europe and America, upon the prognosis of epilepsy, states that sex plays little part; that the influence of hereditary disposition is important only in the case of preceding epilepsy and insanity; that as to age, cases beginning before 10 years have a bad outlook and cases beginning during the period of puberty show the greatest percentage of recoveries. The earlier a case is brought under systematic treatment, the more hopeful the prognosis. There is greater prospect of arrest of the disease or improvement during the first five than during the second five years of the disease. As regards the frequency of the seizures, it may be stated that the longer the interval between attacks, the greater the prospect of arrest or improvement. The kind of the attack modifies the prognosis, the greatest percentage of cures being in cases of *petit mal*. The question as to whether there is a cure of epilepsy may be answered affirmatively. If a case is arrested for nine years, it may be regarded as cured.

Treatment. (a) *Surgical.* Spratling and Roswell Park[3] discuss the subject of bilateral cervical sympathectomy for the relief of epilepsy, and they report three of their own cases. Winter in 1902 collected 213 cases of such operation, the results being fairly encouraging. The excuse for the operation is a double one: first, by cutting off a certain amount of sensory irritation from the viscera, thus quieting the cervical cortex by relieving it of work; and, secondly, by influencing directly the circulation of blood in the cerebral vessels the vasomotor control being through the cervical sympathetic fibers. Physiologic experiments on rabbits, conducted by Lafforgue prove that spasmodic

(1) Journal of Nervous and Mental Diseases, 1905, p. 321.
(2) Edinburgh Med. Journal, December 1904. (Same article in Boston Med. & Surg. Jour., Feb. 16. 1905.)
(3) Journal of Nervous and Mental Diseases, April, 1905.

ischemia of the whole cerebrum, cerebellum and medulla oblongata follows stimulation of the proximal stump of the second cervical sympathetic nerve. Jonnesco, basing his conclusions on his own operations, of which he had done 96 up to the time of Winter's article, concludes that the cervical sympathetic nerve contains vaso-constrictor fibers for the head, face and heart; and vaso-dilator fibers for the brain. The 3 cases operated on by Roswell Park are carefully detailed, together with the technic of operation. Examination of the excised nerves showed pathologic changes in each case.

(b) *Medical.* H. Gerhartz, Jr.[1] discusses the *serum treatment* for epilepsy, advocated by Ceni (mention of which was made in a former volume of the Practical Medicine Series). The author's conclusions are based upon 10 injections each in two cases. He believes that the serum treatment has no effect whatever upon the number of the convulsions. [The serum treatment may now be considered as relegated to the past.—ED.]

Salt Withdrawal in Epilepsy. Hoppe[1] calls attention to the law that the addition of new gas has no influence upon the dissociation of the atoms of the original gas, when the new gas contains no dissociation products of the original gas; and he affirms that the same law holds true in solutions. If two salts contain ions in common, they influence their mutual dissociation. Thus the addition of chlorid of mercury to bichlorid of mercury greatly lessens the bactericidal power of the latter. Thus, too, it is irrational to prescribe a mixture of halogens combined with alkaline bases, such as the bromid and the iodid of potash, because the number of active molecules is greatly reduced. For this reason, if to a solution of sodic bromid there be added a solution of sodic chlorid, the dissociation of the bromin atoms will be much reduced. The greater, then, the reduction of the chlorid of soda in the food of a patient taking sodic bromid, the more effective the latter will be, and the less sodic bromid will be needed. Hence, there is reason in the Toulouse-Richet teaching of salt abstraction in epilepsy while sodic bromid is given.

Of the three most commonly used bromid preparations

(1) Neurolog. Centralblatt, Sept. 16, 1904.
(2) Ibid., Nov. 16, 1904.

(sodic and potassic bromid, and bromalin) bromid best raises the osmotic pressure in the blood. Careful experiments made by Hoppe upon epileptics, the basis of determination being alterations in the freezing-point of the urine, demonstrated that the potassic salt makes the greatest demands of the three upon the eliminating organs. Hence, if the latter are diseased the sodic salts are much to be preferred. On the other hand, the largest proportion of bromid salts remains in the blood-serum, and if it be not promptly eliminated, it is deposited in brain and kidneys, quickly leading to signs of bromin intoxication.

W. A. Turner[1] subjected 8 cases of epilepsy to a regime of salt abstraction during the administration of bromid, and he gives a detailed report of them. He concludes that there are cases of confirmed epilepsy in which the number of attacks diminishes while the treatment is followed, and that there are other milder cases in whom the improvement lasts even after the treatment is stopped. It is especially useful in cases where there is a lack of toleration for bromids. Turner believes that the Toulouse-Richet method may at times be of great service in particular cases. He again alludes to salt starvation cure or dechloridation in *The Lancet*.[2]

L. J. J. Muskens,[3] in a lengthy article upon the treatment of epilepsy, particularly by dechloridation, concludes in effect as follows: "(1) Dechloridation has apparently a greater influence upon the efficiency of the bromin than upon the acne production. The latter seems caused wholly by the amount of bromid salts stored up in the body, rather than by the number of bromin-ions circulating in the blood. (2) Men are much more resistant than women and endure the treatment better. Age has slight influence. (3) *Grand mal* is more easily influenced than *petit mal,* and the psychic equivalents less than either of the two former. (4) Indications that the patient is standing the treatment unsatisfactorily are dizziness, weakness in the legs, thick tongue and speech, constipation in the old, diarrhea in the young, morbid mental weakness, and sometimes, especially in older persons, edema, particularly in the face." (His remaining conclusions are of lesser importance.)

(1) Review of Neurology and Psychiatry, December, 1904.
(2) March 18, 1905.
(3) Neurolog. Centralblatt, March 1, 1905.

... of the hypochlorization ... carefully studied, and concludes ... chlorization method controls the ... about one-half the amount of ... has little or no effect on the ... is apt to cause consti- ... fail to satisfy the pa- ... success by intelligent ... the middle grade of ... the will power ... poor diet, in ... sodium bro- ... advantage with ... can be con- ... cure.

... to this ... physicians ... neurologists ... treatment upon ... and ... long-con- ... patients, ac- ... left ... and ... pos- ... is that ... from ... of ... of the ... muscle. This seems ... other because ... When one has ... most compressed ... the bulk of ... explanatory hypothesis ... directly ... ease in sur- ... ud.

S. G. T...

... has potential

(1) Boston ...
(2) Rev. ... Nov 20, 1902.
(3) N. Y. ... Ind. Medical ... 1902.

currents, and x-radiations in the treatment of epilepsy. He believes that the effect of these forms of energy is to aid the nutrition of the brain, restoring sensory and motor centers to their normal functions. The patient is treated every other day, beginning with x-radiation for five to ten minutes from a high tube, placed 6 to 10 inches above the head, so that the rays strike directly upon the interior and occipital parts of the brain. Then the patient is subjected to a high frequency current applied over the brain for ten minutes and over the spine for five minutes. Small doses of bromids (15 to 60 grains per diem) were also given. He regards 25 per cent of his cases of *petit mal* as tentatively cured, 20 per cent of the Jacksonian epilepsy, and 12 per cent of *grand mal* as cured. All cases were more or less improved, both in respect to the seizure and also its severity. The general mental and physical condition was also much improved. [None of these cases have been followed as yet a whole year.—ED.]

Allen McLane Hamilton[1] discusses the treatment of epilepsy in connection with auto- and hetero-toxins, after having condemned the indiscriminate use of bromids. He has for some years been much impressed by the fact that toxemia enters much more largely than is generally supposed in the pathogeny of epilepsy. As Voisin has pointed out, there is a general resemblance between the epileptic attack and certain ordinary forms of poisoning of the central nervous system. Indeed, the occurrence of the fit betokens the accumulation in the body of some particular toxic agent. The condition of the blood, urine and circulation are those common to many forms of toxemia. This autotoxemia may occur in three different ways, each requiring its own appropriate treatment: (1) The toxemia may be of gastrointestinal origin, as described by Bouchard, and by Herter and Smith. There are quantitative changes in the uric acid and xanthin; and toxic substances are absorbed from the intestines during digestion, formed at times by foreign bacteria. (2) The toxemia may be bacterial, due to the introduction of certain familiar cocci into the blood. (3) The toxemia may be due to the formation of a toxic substance in the body, such as cholin,

(1) Medical Record, Dec. 3, 1904.

first described at length by Mott, and later by Donath. In fact, these observers have established the law that the process of nerve-degeneration comprises the disintegration of lecithin, and liberation of the phosphorized portion of the molecule, and the formation of cholin.

Evidently the proper therapeutic measures are those which will do away with the toxic state. Hence, Hamilton advocates: (1) hydrotherapy, carbonic-acid baths, friction, passive exercises, walking and gymnastics for the purpose of eliminating waste or toxic products. The diet should be carefully regulated according to the idiosyncrasies of the individual, and nitrogenous food interdicted as much as possible. No large quantity of food should be taken at any one time. (2) If intestinal autotoxis exists, cholagogs and appropriate ferments, as well as antiseptics, should be prescribed. (3) Everything possible should be done to prevent the lighting up of gross intracerebral pathologic processes, with the resulting formation of cholin. The equilibrium of the arterial pressure should be everywhere maintained. (4) Bromid should be given only in doses sufficient to diminish the activity of the cortical motor cells.

Peter Rixen[1] advocates the use of neuronal, first introduced to the profession as a hypnotic by E. Schultze of Bonn. Neuronal, being a bromidethylacetamid, is naturally suited for the treatment of epilepsy, and Rixen tried it on 80 epileptic female patients in the Berlin Hospital for Epileptics. It was found of more value in conditions of excitement, motor unrest, and delirious states than in convulsions; and it was given in doses of 3 to 4 grams (45 to 60 grains) daily. Owing to its hypnotic effect, larger doses cannot be given. It proved especially useful in postepileptic states, especially headache and nervous menstrual disorders. No unpleasant results followed its use.

Treatment of Status Epilepticus. Morton[2] describes a method of treatment in use the past year at the Massachusetts Hospital for Epileptics. A solution of sodic bromid, of a strength of 30 grains to the ounce, rendered sterile, is injected in doses of from 2 to 3 ounces in the back just below the angle of the scapula. Sometimes as much as

(1) Muench. med. Wochenschrift, Nov. 29, 1904.
(2) Boston Medical and Surgical Journal, June 15, 1905.

6 ounces of solution have been found necessary. There need be no fear of abscess formation in this locality. The earlier the injections are commenced in *status epilepticus,* the better. Indeed, the solution seems to have great prophylactic value, preventing or aborting threatened attacks. If any patient in the institution has two convulsions in succession, the injection is at once given, ten hypodermics of 20 minims each being administered. If the patient continues to have convulsions, ten such hypodermic injections are given after each convulsion, till he has received forty. If this does not suffice, then 1 to 4 ounces of the sterilized bromid solution are given with a big antitoxin needle in the back.

Lumbar puncture has been tried, with indifferent success, in 7 cases, 3 dying and 4 recovering. It was used only when the bromid given hypodermically had failed. The 4 cases which recovered showed marked improvement after the withdrawal of 10 to 15 c.c. of cerebrospinal fluid. In one case 10 c.c. of the sterilized bromid solution was injected into the subdural space, after the removal of 20 c.c. of cerebrospinal fluid, the patient having only one convulsion in the next fifteen hours.

Tremaine[1] reports 2 cases of status epilepticus treated successfully at the Craig Colony by venesection and intravenous injection of normal salt solution; about eight or ten ounces of blood being withdrawn, and a quart to three pints of fluid injected. Their early use is advocated.

Conrad Alt,[2] in a very long and thorough review of the treatment of status epilepticus, advocates iodid of potash as an efficient remedy. Often colonic flushing is of value, or drugs, such as bromid, chloral hydrate, amyl hydrate, dormiol, or opium, may be given well diluted by rectum. Chloroform is also a valuable remedy. Hydrotherapy, venesection, and intravenous saline injections are all useful.

(1) N. Y. Med. Jour. and Phila. Med. Jour., Sept. 10, 1904.
(2) Muench. med. Wochenschrift, March 28, 1905.

MIGRAINE.

Pathologic Seat. Leopold Levi[1] declares that migrane is characterized essentially by hemicrania, associated with a group of other phenomena, chief of which are nausea, vomiting, vertigo, vaso-motor and secretory disturbances, including ptyalism and polyuria, and the oculo-pupillary syndrome. Dubois-Raymond regarded it as due to a disturbance of the sympathetic system, and Brissaud has looked upon it as a form of trigeminal neuralgia. Levi regards it rather as having its seat in the floor of the fourth ventricle, for by this hypothesis all of its symptoms are best explained. Migraine, then, is to be looked upon as a bulbo-pontine syndrome of variable etiology. Ophthalmic migraine may find its explanation in the connection between the descending motor root of the fifth nerve and the quadrigeminal bodies.

Ophthalmic Migraine. Meige[2] reports a case of ophthalmic migraine associated with transitory hemianopia and aphasia, photophobia and a winking tic, in a patient of 73 years of age. In addition, there was paresis of the right side of the face and following the attack was somnolence. Meige attributes the site of the disturbance to the medulla, and regards an angiospasm as the cause. Kollaritz[3] reports the case of a girl of 17 years, without hereditary taint, who, from the age of 7 years, had such headaches followed for several days by oculo-motor paralysis, hyperesthesia in the ophthalmic division of the trigeminal nerve, and amaurosis of the right eye. The paresis of the oculomotor nerve, and the amblyopia of the right eye, persisted between attacks. They are regarded by Kollaritz as the result of the attacks.

Otic Migraine. Escat[4] affirms that there is an otic migraine, the homologue of ophthalmic migraine. In this form disturbances of hearing are observed rather than ophthalmic disturbances; but sometimes both sets of disturbances occur in the same patient.

Treatment. P. Consiglio[5] reports the case of a woman

(1) Revue Neurologique, February 15, 1905.
(2) Revue Neurologique, Sept. 30, 1904 (also Le Progrès Medical, 1904, p. 125).
(3) Deutsche Zeitschrift für Nervenheilkunde, 1904, p. 128.
(4) Revue Neurologique, Dec. 15, 1904.
(5) Gazetta degli Ospedali e delle Cliniche, Nov. 20, 1904.

of 40 years, in whom the attacks were always associated with the menstrual period, the disease beginning with the establishment of the menstrual function. It therefore occurred to Consiglio that possibly the migraine was due to a utero-ovarian toxemia, possibly resulting from transitory thyroideal insufficiency. Hence, thyroid extract was given daily in the form of two Burroughs-Welcome tablets, and three daily just before the expected menstruation. The migraine disappeared. When the extract was stopped the migraine began again. The difficulty with this method of treatment is palpitation of the heart and loss of weight.

CHOREA.

Pathology: Spiller,[1] reviewing the subject of choreiform movements, calls attention to the so-called "chorea bodies" found in many cases. Hudovernig[2] has found that encephalitis and meningitis are common. His conclusions are as follows: "Chorea minor is an infectious disease and the infection is hematogenous. In the mild cases the infection causes a disturbance of nutrition, but in more severe cases it causes changes in the blood vessels and formation of colloid bodies, the latter being an evidence of a severe form of the disease. Chorea bodies are characteristic of chorea, although they are not found in every case. Choreiform movements are always the expression of direct or indirect irritation of the pyramidal tracts at some part of their course."

Symptoms. Meige[3] calls attention to hippus in chorea. Hippus, an oscillation taking place in the size of the pupil, when it is subjected to extremes of illumination, before it reaches equilibrium, is observed in many nervous diseases, such as tuberculous meningitis. Indeed, even in an unchanging light, the pupil may alternately dilate and contract, as took place in a case of cerebellar tumor reported by Meige in 1903. In Sydenham's chorea hippus is a fairly constant phenomenon, though hitherto it has not been mentioned as occurring in that disease. In 7 cases

(1) Journal of the Am. Med. Assn., Feb. 11, 1905.
(2) Archiv für Psychiatrie, XXXVII, p. 86.
(3) Revue Neurologique, Nov. 30, 1904.

examined, it was uniformly found. On the other hand, in a case of generalized tic it was absent.

Babinski[1] has met with the sign known as "combined flexion of thigh and trunk" in quite a large number of cases of Sydenham's chorea. It was especially noted in cases of hemichorea, being present on the side on which the involuntary movements predominated. As a rule, the sign disappears with disappearance of the chorea. This sign is usually one of the most common of the objective ones in organic hemiplegia, but absent in the hysterical form. It therefore serves to bolster up the conclusion that chorea is the result of interference with the function of the pyramidal tracts. This sign will furthermore serve to differentiate hysterical chorea from Sydenham's chorea.

Michel[2] differentiates sharply between *chorea mollis* and the paralyses of chorea. In the former the paralyses, by reason of their intensity and wide-spread distribution, comprise the main symptom, the choreic movements being merely an accessory symptom. In the cases of "paralyses of chorea," the paralyses are merely an episode in the chorea; the movements constitute the essential symptom, the paralyses being accessory. There are certain analogies, however, between chorea mollis and the paralyses of chorea. In each the paralysis is flaccid, there are no sensory disturbances, and both tend toward recovery. In chorea mollis, the paralyses are insidious in origin, usually involve all four extremities, the muscles of the neck and larynx, and they recede slowly. The paralyses of chorea may come on before, during or after the chorea, and as a rule are mono-, hemi-, or paraplegic. Chorea mollis is probably due to dynamic disturbances of the large motor cells of the pons, medulla, and cord, and doubtless has some relation to asthenic bulbar paralysis.

Mental Disturbances in Chorea. Ruppel[3] enumerates the mental symptoms as follows: (a) Elementary psychic disturbances: excitement, irritability, fear, a tendency toward being notional, fleeting visual hallucinations, terrifying dreams; (b) momentary and mild febrile delirium; (c) psychic exhaustion phenomena: forgetfulness, inabil-

(1) Revue Neurologique, Jan. 30, 1905.
(2) Ibid., Dec. 30, 1904.
(3) Muench. med. Wochenschrift, March 17, 1905.

ity to concentrate, mental abstraction, indifference, mental confusion even to the point of an exhaustion-delirium because of insomnia, restlessness, defective nutrition, fever; (d) mental disease proper, due to the same source of infection as caused the chorea, such as simple or hallucinatory confusional insanity and even stupor or dementia; (e) complicating psychoses, due to the same psychic trauma as causes the chorea, or independent of it.

Ruppel carefully recounts a case in point, in which the differential diagnosis between Sydenham's and Huntingdon's chorea was doubtful. Another case is mentioned, of a girl of 22 years, with Sydenham's chorea, in whom visual hallucinations occurred in the night.

Hoisholt[1] reports 2 cases of dementia occurring in Huntingdon's chorea. In chorea minor (Sydenham's) the mental implication is usually not grave. It is seldom more than a condition of more or less marked fatuity—rarely a real psychosis. The character of the child affected becomes changed; there are lapses in the moral sense, mendacity, perversity, emotional outbursts, and irritability. Disturbances of memory and of the faculty of attention are also common. In Huntingdon's chorea the patients become forgetful, weak in judgment, irritable and wavering, incapable of regular activity. Now and then sensory hallucinations or percutory ideas are detected. Ultimately the mental and emotional activities are annihilated and the patient lapses into a condition of dementia.

Familial Chorea. Brissaud, Rathery and Bauer[2] presented at the March meeting of the Société de Neurologie of Paris two sisters with chorea, of a family of four children, all of whom have or have had chorea. In these cases the chorea was looked upon as most likely epidemic.

Treatment. Spiller[3] regards confinement to bed as of the greatest importance in severe cases. When Fowler's solution is given its effect should be carefully watched. Spiller usually orders only 2 fluid drams at a time, directing that the prescription be not renewed without permission. Spiller does not recommend the salicylic acid treatment. Riviere[4] has treated 35 cases of chorea by ergot and

(1) Am. Jour. Med. Sciences, Jan., 1905, p. 77.
(2) Revue Neurologique, March 30, 1905.
(3) Journal of the Am. Med. Assn., Feb. 11, 1905.
(4) British Medical Journal, Feb. 18, 1905.

arsenic. The ergot was given in 30 to 60 minim doses of the fluid extract, three times daily, with two minims of the British liquor strychninæ. In about half of the cases the ergot has a marked curative effect; in the others the disease was not influenced. In the latter cases arsenic was then substituted. Ergot and arsenic were also used in combination, a mixture being given consisting of 60 minims of fluid extract of ergot with 3 minims of liquor potassii arsenitis. This is very effectual in all cases. Hollopeter[1] emphasizes the virtues of *prolonged warm baths* in shortening the attacks of chorea. The child is placed in a bath with a temperature of 90 to 98 degrees, and entertained by the nurse or mother by floating toys or other devices. The whole body except the head and neck is kept immersed for at least one or two hours, twice daily.

Huntingdon's Chorea. Spiller[2] calls attention to the gross cerebral lesions of Huntingdon's chorea and their superficial resemblance to those of paralytic dementia. In a case examined by Spiller, small collections of round nuclei were seen about some of the capillaries of the cortex of the paracentral lobule, and also elsewhere in the cortex not about the vessels. Malcolm Mackay[3] reports 3 cases of hereditary chorea in a family in which there have been altogether 18 cases. Weyrauch[4] reports 2 cases from a family, male cousins. In all, 8 members of this family had been victims of Huntingdon's chorea, 5 having died of it. In 6 of the cases, where the time of onset was known, it was between the 30th and 43rd years. Vaschide and Vurpas[5] report cases where the movements ceased abruptly a few days before death, contrary to the rule. Post mortem examination showed the cause to be a recent diffuse menigo-encephalitis involving the whole central nervous system.

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(1) Journal of the Am. Med. Assn., Dec. 31, 1904.
(2) Journal Am. Med. Assn., Feb. 1, 1905.
(3) Medical News, Sept. 10, 1904.
(4) Muench. med. Wochenschrift, Feb. 7, 1905.
(5) Revue de Med., Sept., 1904.

DISEASES OF THE BRAIN.

Localization. Arthur P. Herring[1] reviews the most recent views concerning localization. Each hemisphere in a general way is made up of three special areas: (1) the higher psychic, abstract concept area of Mills, or the frontal association area of Flechsig; (2) an intermediary region of concrete concept of Mills, with its numerous subdivisions, or Flechsig's parieto-temporo-occipital association area; (3) the special sense and motor areas, as vision, audition, motion, etc. The first two areas naturally are connected only with association-fibers, while the third area is connected with projection-fibers connecting it with the periphery. The accompany figures from Mills are fitting in this connection.

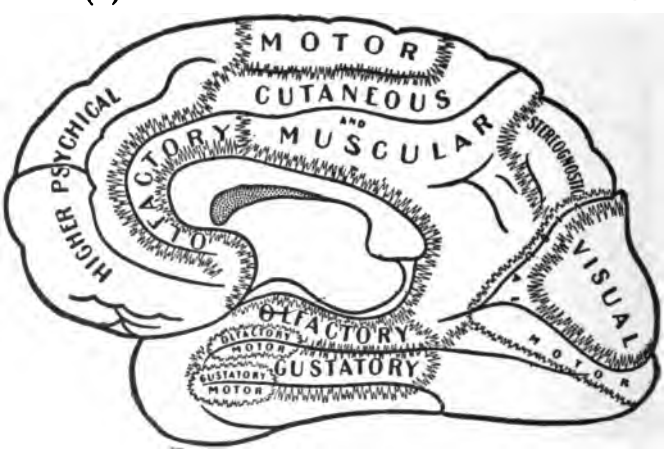
CONCRETE CONCEPT.

Areas and centres of the lateral aspect of the human hemicerebrum. Motor area is placed *anterior* to the Rolandic fissure. Diagram by C. K. Mills, M. D., from the University of Pennsylvania Medical Bulletin, May, 1904.

Until recently, owing to the teaching of Ferrier, the motor cortex has been said to lie both in front and behind the fissure of Rolando, but later study by Mills, Flechsig, Grünbaum and Sherrington indicates that the motor area

(1) Medical Digest, October, 1904, p. 1.

lies entirely in front of the Rolandic fissure, except for a bit of the Rolandic floor. It will be noted that in the diagrams of Mills the motor cortex lies in front of the Rolandic fissure. Herring's conclusions follow: "(1) The psychic function of the left prefrontal lobes is pretty definitely determined. (2) The motor area is placed anterior to the central fissure in the precentral convolution and upper part of the paracentral convolution, there being a specific type of motor cortex. (3) The motor speech center in the posterior part of the inferior frontal convolutions extends probably back to the anterior portion of the insula. (4) The position of the special sense areas, as the gustatory, olfactory, visual, and auditory, remains the same. (5) The various and manifold subdivisions of the



Areas and centers of the mesial aspect of the human heml-cerebellum. Diagram by C. K. Mills, M. D., from the University of Pennsylvania Medical Bulletin, May 4, 1904.

association-area as shown in the Mills diagram are still *sub judice.*"

Hubert M. Turnbull[1] reports a remarkable case of bilateral loss of the postcranial cortex, apparently congenital in a girl 24 years of age, who died from severe burns. In life the girl was of course blind, there being no occipital cortex. She was late in uttering recognizable speech, but her

(1) Brain, Summer 1904, p. 209.

range gradually and progressively increased. She could answer some questions sensibly. As a rule she alluded to herself only in the third person. Her hearing was acute. She evidently had no tactile sense or tactile memories. She never felt of anything. Her sense of smell was fairly acute. She would smile when pleased, but she never laughed heartily.

Most important as bearing on the location of the motor function is the fact that there were no pareses or paralyses. This fact speaks strongly for the localization of the motor functions wholly in the precentral gyrus. However, she had some contractures, the wrists being strongly flexed and adducted, the fingers flexed, and the feet in equino-varus. The author has worked out the whole pathologic anatomy very thoroughly.

C. Burt[1] also publishes a case of a lesion of the post-central gyrus without paralysis. Post mortem examination showed a small hyperemic softened area in what is usually regarded as the arm center. In life there was absolutely no paralysis. There were two convulsions, however, death taking place in the second.

W. B. Warrington[2] reported a case of thrombosis of the left middle cerebral artery, with cortical softening and total aphasia without loss of consciousness. In this case, a man aged 70, in spite of complete paralysis of the face and upper limbs on one side, the postcentral gyrus was entirely free from disease. [Here, again, is a case confirming the claims of Grünbaum, Sherrington, Mills and others.—Ed.]

Chas. K. Mills[3] discusses at length his so-called "concrete concept area," by which he means the parieto-temporo-occipital region which lies between the projection-areas of the cortex and largely corresponds to the great posterior association-area of Flechsig. The subdivisions of this area include the stereognostic perception, word-seeing, letter-seeing, number-seeing, object-seeing, orientation, naming, object-hearing, word-hearing, and intonation. Possibly the muscular sense should be here included, but at present Mills puts it in the primordial regions.

(1) British Medical Journal, March 12, 1904.
(2) Liverpool Medico-Chirurgical Journal, 1903-04, p. 227.
(3) Medical News, Nov. 5, 1904.

"In order that object-blindness shall be complete and permanent, the recorded cases would seem to show that it is necessary for lesions to be present in somewhat corresponding locations in both hemicerebrums. Transient or recurrent object-blindness is, however, sometimes exhibited by patients with a lesion confined to one hemisphere." The center for object recognition by sight is in the temporo-occipital convolution from the second to the fourth or fifth gyrus.

The question of the existence of separate centers for color, luminosity and form is unsettled. If special centers for color exist, they are probably just within or just outside of the primary visual areas for light; in other words, within or on the borders of the cuneus and the first occipital convolution. A few cases of unilateral congenital color blindness indicate the existence of special localization for color representation. Cases of hemiachromatopsia without hemianopia are also extant. Two cases of such hemiachromatopsia are recounted, both with post-mortem examinations. In the first the lesion was found at the apex of the left cuneus and the adjoining upper part of the lingual convolution. In the second there was a recent large hemorrhage in the right hemisphere, destroying the centrum ovale and the basal ganglia, and filling the right lateral ventricle; and a fresh hemorhage, about the size of a bean, situated at about the junction of the anterior and middle third of the left corpus callosum. Parts of the white substance of the third occipital convolutions and the occipital ends of the lingual and fusiform convolutions, together with the posterior inferior point of the cuneus were destroyed. These two cases prove that a center for color recognition, and for chromatic sense, lies on the mesotentorial surface of the hemicerebrum, probably in the fusiform and lingual convolutions; very likely it also extends into the adjoining portion of the cuneus. It is more than probable that color-recognition has two distinct cortical areas of centers of representation, one mesotentorial for the chromatic half-fields and the other lateral occipital in the neighborhood of the angular gyrus for color recognition by the macula.

The cerebral representation of the musical sense, or intonation, or note-hearing, appears to be entirely separated

from that for word-hearing, although doubtless it is not very far removed from the latter. Mills places it in the cephalic extremities of the first and second temporal gyri, where its location is indicated by anatomic, morphologic and clinico-pathologic investigations. Some aphasic patients may retain the musical faculty. A few cases, too, of note-deafness without word-deafness have been recorded. On the other hand, note-deafness and word-deafness have been found together in the same case in a large number of instances. Evidently, then, while the center for tones is separate from that for words, the two are in close propinquity. "In the few well observed and well recorded cases of amnesia in which necropsies have been obtained, the lesions have involved the anterior portion of the temporal lobe, usually the first or the first and second temporal gyri, although sometimes in addition some of the cortical matter hidden within the Sylvian fossa."

The Location of the Word-Hearing Center. W. G. Spiller and H. B. Allyn[1] reported at the Philadelphia Neurological Society the case of a man of 57 years, right-handed, who in life could understand all that was said to him, could relate anecdotes, and who showed very few symptoms of aphasia. Nevertheless, post mortem examination showed complete destruction of the first left temporal convolution. The second was well developed. Evidently we must assume that the retention of the function of word-hearing was due to the preservation of the latter convolution, or else was due to an unusual development of the first right temporal convolution. In the discussion Mills stated it as his belief that the lower portion of the hinder part of the first temporal convolution and the upper part of the second temporal convolution are concerned in word-hearing. Inasmuch as in Spiller's case there was no involution of the left inferior frontal convolution, it seemed probable to him that the man's word-centers were developed on the right side.

The Location of the Visual Function. Beevor and Collier[2] report a case of quadrantic hemianopia in a man of 55 years. Up to the time of Beevor and Collier's case, but one case of quadrantic hemianopia from a cortical lesion

(1) Journal of Nervous and Mental Diseases, March, 1905.
(2) Brain, Summer 1904, p. 153-162 (with bibliography).

had been reported with pathologic examination, and even in this one the microscopic examination was omitted. Other cases reported have merely afforded evidence that the dorsal part of the visual cortex and the dorsal portion of the optic radiation between this and the external geniculate body are concerned with the lower retinal quadrants, and that the ventral portions of both visual cortex and optic radiations are concerned with the upper quadrants. The patient, long under observation, showed as a sole manifestation of gross nervous disease, blindness of the left upper quadrants of both visual fields, the fixation-point escaping. Post mortem, there was found an occlusive lesion of the right posterior calcarine artery which had caused destruction: (1) of the right fusiform lobe for its posterior .8 inch. (2) of the right lingual lobe from the junction of the calcarine and parieto-occipital fissures to the pole of the hemisphere, (3) of the whole cortex in the depth of the calcarine fissure, (4) of the greater part of the inferior cuneal gyrus, small areas only at the anterior and posterior limits of this gyrus being free. The necrosis did not involve the optic radiation at any point. The only parts of the cortex of the mesial aspect of the occipital lobe which had escaped destruction were the upper two-thirds of the cuneus and the anterior and ventral portion of the fusiform lobe. The lower quadrants of the visual fields were entirely unaffected, and, until it was pointed out to him, the patient was unaware of the visual defect. The authors submit, therefore, that this case affords conclusive evidence that the cortex of the upper two-thirds of the cuneus is the primary center for the lower quadrants. The primary half-vision center is considered by von Monakow to occupy the cortex of: (1) the entire lingual lobe behind the junction of the calcarine and parieto-occipital fissures, (2) the entire cuneus and extending for some ½ inch or more on to the external aspect of the occipital lobe. He thinks that the calcarine fissure probably forms the line of separation of the representation of upper and lower quadrants. According to the authors, this case strongly supports von Monakow's localization of the half-vision center.

The conclusions of von Bechterew[1] concerning the cortical center of vision follow: Destruction, whether circum-

(1) Monatsschrift für Psychiatrie und Neurologie, X, Heft 6.

scribed or extensive, of the cortex of the dorsolateral portions of the occipital lobe is always accompanied by homonymous hemianopia, bilateral, but more marked in the eye of the opposite side. In circumscribed lesions hemiamblyopia occurs rather than hemianopia. The duration of the disturbance is variable, according to the extent of the lesion; the disturbance progressively diminishes, more slowly in the eye of the opposite side, and may completely disappear. After a cure, a new lesion in the cortex in the neighborhood of the old one will produce a new hemianopia. In very extensive lesions of the hemisphere there seems to occur, simultaneously with the double homonymous hemianopia, an amblyopia of the opposite side, which would indicate the presence in the cortex of the zones superimposed over a great extent, one in relation with the corresponding halves of the two retinæ, the other with the retina of the opposite side. Lesions of the mesial surface of the occipital lobe also produce a double homonymous hemianopia.

H. T. Patrick[1] reported a peculiar case of lack of orientation and occipital lobe disease, without post mortem examination, however. The patient, a man 53 years of age, attributed the onset of his trouble to a severe cold following exposure, the main feature being severe pain in the back of the head and back of the neck, followed by two or three weeks of terrific occipital headache. Examination showed: (1) greatly restricted visual fields, due to practically a homonymous hemianopia with the additional loss of the upper quadrant of the hemianopic field; (2) impairment of the sense of orientation; (3) impairment of visual memory, especially for localities, location and directions. His greatest practical difficulty was in locating himself in reference to his immediate environment. The conclusion was drawn that a focal encephalitis involved both the right and left occipital lobes, chiefly of the lower part of the cuneus and the lingual lobe, in accordance with Beevor and Collier's article to which reference was just made. In the discussion following, L. F. Barker stated that the lesion must involve the entire occipital region, according to von Monakow.

(1) Journal of Nervous and Mental Diseases, 1905, p. 388.

The Location of the Motor Speech Function. Chas. K. Mills[1] assigns to this function the hinder portion of the subfrontal or third frontal gyrus, and the insula or at least its anterior half. A patient is cited showing in life verbal amnesia and general disorganization of the motor speech function in whom, after death, the lesion involved the anterior half of the insula. E. A. Spitzka holds that probably only the preinsula is concerned in speech, being an association-area uniting the sensory side of the zone of language or a portion of it with the third inferior frontal gyrus. Mills holds that Broca's convolution and the preinsula conjointly constitute the motor speech area. In this area language is organized for the use of the executive centers concerned with speech; in other words, for the facial, lingual, and laryngeal centers close at hand. Short association-fibers connect these two areas. Mills supports this claim by a case first reported in 1889, in whom articulation-defects caused by a lingual, oral, and slight facial paresis were present, while speech was entirely unaffected except for its mechanical execution, the lesion being found postmortem in the lower extremities of the central convolutions both on the lateral and Sylvian surfaces, and a spot ½ inch in diameter about the middle of the internal portion of the insula. Moreover, Mills has shown in one case that faradization of the subfrontal area during an operation gave negative results, whereas in the same case faradization of the centers for the face, eyelids, and head gave prompt and positive response.

W. C. Krauss[2] reports an interesting case of *contre-coup* hemorrhage exactly over the third anterior frontal gyrus of the left hemisphere. The hemorrhage was slowly progressive, as no difficulty of speech was noticed until some sixty hours after the injury, producing symptoms of aphasia and paraphasia. The area invaded by the hemorrhage extended downward and backward, aided by gravity, reaching the facial center, producing facial Jacksonian epilepsy on the fourth day, on which he was operated. A prompt recovery followed.

Senator[3] reports a case of aphasia occurring in a right-

(1) Am. Jour. of the Med. Sciences, September, 1904.
(2) Ibid., September, 1904.
(3) Neurolog. Centralblatt, Oct. 1, 1904.

handed person, associated with a *left-sided* hemiplegia, turning of the head to the left, and conjugate deviation of the eyes to the left. In addition, there was some word-deafness, and agraphia and alexia. Postmortem examination showed extensive softening, due to embolism, of the right hemisphere, especially of the temporal lobe and the area about the right Sylvan fissure. In this patient it would seem that the whole speech area, both motor and sensory, was located in the right cerebral hemisphere.

The Location of the Word-Writing Center. Chas. K. Mills[1] states that he has long believed in the existence of a separate graphic or writing-center, first located by Exner in the posterior end of the second frontal convolution, but its existence being later denied by Byrom Bramwell, Déjerine, and others. In support of his view Mills reports illustrative cases which he divides into two groups: (1) cases in which a lesion is absolutely circumscribed to Broca's convolution, or the insula, or both, with motor aphasia and without motor agraphia; (2) cases in which a lesion is absolutely circumscribed to the posterior end of the second frontal convolution, with agraphia and without motor aphasia. A case cited, seen with McConnell, Spiller and Frazier, is detailed. All attempts to write, either spontaneously or from dictation, were absolutely futile, although the patient held and handled his pen and pencil properly and copied a sentence with comparative ease. There was in no sense an aphasia such as results from a lesion of the inferior frontal convolution or the insula. The tumor found lay athwart the second frontal gyrus, exactly over the cortical area customarily assigned to Exner's writing-center.

APHASIA.

Word-Blindness. M. A. Halipre[2] reported at the December meeting of the Société de Neurologie of Paris the case of a woman of 67 years showing as her chief defect autonomasia or anomia (the inability to name objects), and word-blindness. In addition, she showed paraphemia. "I would

(1) Am. Jour. of Med. Sciences, September, 1904.
(2) Revue Neurologique, Dec. 30, 1904. Also, Nouvelle Iconographie de la Salpêtrière, Vol. 18, p. 36.

like to say, but I cannot," was her only answer to anything and everything. She could count to 6. She articulated well, showed no word-deafness nor mind-blindness, though she showed complete word-blindness. She could not read her own name, but she could pick out isolated letters. After a few months she recovered. Death taking place three years later, an autopsy revealed an old softening in the posterior portion of the subparietal gyrus, involving the gyrus supramarginalis, the gyrus angularis, and the third occipital gyrus in the left hemisphere. It extended to within .8 inch of the tip of the occipital lobe. The underlying medullary substance was also involved, chiefly the fibers of the superior longitudinal association bundle.

Levi and Tagnet[1] report a case of pure word-blindness associated with right lateral homonymous hemianopia and locate the lesion in the visual center, stating that it also involves the association fibers leading to the gyrus angularis. [This is the standard teaching.—ED.] The authors attributed the involvement of these fibers to edema, but in the discusion Brissaud chose rather to refer it to an intoxication.

Déjerine and Thomas[2] report a case of word-blindness associated with agraphia. Attention is called to the fact that two types of word-blindness exist: (1) Word-blindness associated with agraphia, and (2) pure word-blindness of which the case of Levi and Taguet is an example. The former is due to a lesion of the gyrus angularis; the latter to a lesion involving the association fibers running from the cuneus to the angular gyrus. A case is reported of a right hemiplegia with contracture, sensitivo-sensorial hemianesthesia, right homonymous hemianopia, word-blindness, letter-blindness, and agraphia, in a woman 70 years of age. The agraphia was present both in spontaneous writing and writing from dictation.

They[3] also report a case of sensory aphasia in a woman 78 years of age whose brain was carefully examined by the serial section method of examination. The chief symptoms were complete word-deafness, complete word-blindness and

(1) Revue Neurologique, June 30, 1905.
(2) Revue Neurologique, July 15, 1904.
(3) Revue Neurologique, Aug. 15, 1904.

agraphia. In addition there was anomia, an inability to repeat words, and a tendency towards taciturnity. At the autopsy a large focus of softening found in the left parietal lobe and chiefly involving the gyrus angularis completely explained the word-blindness and accounted for the agraphia. Curiously enough, no macroscopic lesions of the posterior third of the second temporal convolution was found to explain the word-deafness. Microscopic examination, however, showed such a lesion, also involving the neighboring portion of the first temporal gyrus.

Two cases of congenital word-blindness are reported by Sidney Stephenson[1] and the recent literature on the subject briefly reviewed. Evidently there exists a congenital condition where the learning of letters or of words from printed or written characters is difficult or even impossible. Of the cases collected by Stephenson 82.25 per cent have been in males ranging from 7 to 23 years of age. The diagnosis offers few difficulties if once the fact be firmly grasped that there is such a thing as congenital inability to read.

AMNESIA.

Marinesco[2] discusses amnesia in an extensive article, confining himself chiefly to motor amnesia, principally instrumental. A case is reported of a man who lost the memory of the movements necessary for playing his instrument. He could not play the simplest airs, although he was capable of executing the most delicate movements with his fingers. Hence, motor instrumental amnesia may exist, independently of any motor or sensory disturbances of the fingers. Vocal amnesia presupposes a lesion of the motor speech-center, in the left hemisphere in right-handed people. Instrumental amnesia presupposes a lesion in *both* hemispheres since both hands are used in instrumental music. Sensory amnesia is of two sorts: (1) Musical alexia or musical blindness in which there is a loss of the ability to read musical notation, and (2) musical deafness in which there is a loss of the ability to identify tunes by the sense of hearing. Many interesting

(1) Lancet, Sept. 17, 1904.
(2) Sémaine Médicale, Feb. 1, 1905.

cases of sensory amusia are also reported. According to Marinesco, the exact seats of the different forms of amnesia remain as yet unknown to us. A very excellent historical summary is found in this review.

The loss of the sign-language in a deaf-mute from cerebral tumor and softening is reported by C. W. Burr,[1] this being the only case which Burr has been able to find after a careful search of the literature. Surely disease of such a nature and so situated as to produce loss of the ability to comprehend and use the sign-language must be very infrequent. At the necropsy the left cerebral hemisphere was found to be a little larger than the right, and its convolutions were flattened on the convex surface, especially in the anterior two-thirds. In the posterior part of the superior frontal gyri was a dark red spot. Beneath was a soft tumor, not encapsulated, surrounded except on its cortical aspect by an area of softening, due partly to hemorrhage and partly to thrombosis. Unfortunately, in a case of these pathologic findings the location of the pantomimic centers cannot be inferred. According to Bastian, amimia and paramimia do not occur in subcortical lesions. According to Mills, lesions affecting the word-hearing centers are more likely to affect pantomime, than lesions affecting the motor speech and writing-centers. Burr remarks that personally he has never seen pure motor aphasia associated with complete amimia. The sign-language is learned by vision, and hence a lesion of the visual center in deaf-mutes would be likely to cause its loss. Whether there can occur a purely motor disturbance of the sign-language is unknown. A pure motor amimia in persons who are not deaf-mutes, unassociated with any form of verbal aphasia, has never been described.

Treatment of Aphasia. Chas. K. Mills[2] reports several cases of improvement of aphasics by careful training.

"The various pedagogic methods of treating aphasics can be summarized as follows:

"1. The method of repetition after others which later becomes that of spontaneous recall as the patient im-

(1) N. Y. Med. Jour. and Phila. Med. Jour., June 3, 1905.
(2) Jour. Am. Med. Assn., Dec. 24, 1904.

proves; and allied or assisting methods like reading aloud, copying and writing from dictation.

"2. Phonetic methods such as the method of the physiologic alphabet suggested by Wyllie and the use of phonetic readers.

"3. The employment of vision to assist in the training as when the aphasic imitates the movements of articulation, enunciation and vocalization as made by others or by himself, in the latter case observing these in a mirror.

"4. The re-training of the patients in the grammar of language when the aphasic is educated and when not, reorganizing so far as possible such language as he originally had.

"5. Various special methods suggested by different authorities, as, for instance, that of Goldscheider of training the patient to repeat meaningless syllables.

"With regard to the method of training by repetition many directions and many illustrations might be given. Those furnished by Dr. C. L. Dana in a recent publication will answer as a basis for work of this kind, and I shall therefore, as they are brief, take the liberty of citing them fully. Such methods can be varied almost indefinitely.

"1. Repeat five exclamatory words, such as: Ah, Oh, or another exclamation expressing joy, anger or other emotion.

"Repeat after the teacher ten single monosyllabic nouns and pronouns.

"Repeat ten polysyllabic nouns.

"Repeat ten verbs.

"In these latter exercises, each time a noun is named, let the patient see the object, feel it, and see the written or printed name of it on a piece of paper before him, thus stimulating his visual, auditory and tactile memories at the same time, as for example: Watch; pencil; pen; cane; box; book; and so on.

"2. Repeat the letters of the alphabet, these letters being held in front of him.

"Repeat the letters of the alphabet after writing and looking at each one.

"Repeat the figures up to ten.

"Repeat while looking at the written figures in front of him. Write and repeat these figures.

"3. Repeat ten simple, qualifying adjectives, such as: White; black; red; smooth; soft; rough. At the same time let him see the object and color, or feel the same.

"4. Later let him try to repeat sentences of three words in which the noun is joined to the adjective, using the familiar nouns and the familiar adjectives already experimented with, thus: Pencil or pen is black; box is white; book is red; and so on.

"5. If the patient ever had any musical capacity, have him sit at the piano and hum the notes of the piano, going through an octave, and then let him try to hum a tune, striking a note at the same time. Finally teach him to sing the tune through and then introduce the possible words. Some patients can sing before they can talk.

"6. Copy sentences made up of the words he is being taught. Let him have an ordinary copy book and have the copy at the top of the page. Let him fill a page every day, trying at the same time to pronounce the words as he writes them. Have him copy first the familiar nouns, and then the simpler verbs, then the simple adjectives; finally let him copy sentences.

"Take a small vocabulary and repeat from this, not trying to enlarge too soon.

"7. Write the letters of the alphabet, and as he writes them, try to repeat them. Do this without a copy, if possible. Then let him write words to dictation, using the same vocabulary above referred to. Finally let him try to write short sentences to dictation, then try to read them after he has written them, with assistance at first, then without.

"8. Write numbers up to twenty and say them out loud when written.

"9. It would do no harm and might be of some benefit to try the effect of hypnotic suggestion in helping him to get along in these exercises.

"10. The patient should allow himself to be read to for a short time twice a day, and he should also try himself to read a quarter of a page every day."

(The article is a very long one and the reader is referred to it for greater detail.)

Influenced by Crocq's observations on the effect of lateral decubitus in epilepsy, Bonnier[1] tried it in a case of a right hemiplegic with aphasia. He could speak plainly when he was upon his right side, but when he turned back to his left or upon his back, he became aphasic again.

DISEASES OF THE MENINGES.

Acute Meningitis. Councilman[2] enumerates as the three organisms most generally causing the disease the diplococcus intracellularis meningitidis, the pneumococcus, and the streptococcus. Epidemic cerebrospinal meningitis is due solely to the first of these, and in the epidemic of 1897 in Massachusetts was found by Councilman, Mallory and Wright in 31 out of 35 cases of the cases coming to autopsy, and in 38 out of 55 cases in which lumbar puncture was performed. It was present in all of the acute cases, but rarely in those which ran a more chronic course. Councilman believes that with rare exceptions all cases of primary meningitis are due to the diplococcus intracellularis.

Purulent Meningitis. Kummell[3] reports a case of suppurative meningitis following fracture of the skull, proved by lumbar puncture. The patient was unconscious and apparently moribund when the skull was opened. Nothing was found other than a very diffuse meningitis without abscess formation. Consciousness returned in six days, and in four weeks the post-operative aphasia disappeared. Six weeks after the operation the patient was dismissed from the hospital.

Tuberculous Meningitis. A cure of a patient has been reported by Rossini[4] who administered subarachnoidean and intravenous injections of iodin. The case was that of a boy of 16 years, and the tuberculous nature of the malady was demonstrated on guinea-pigs. Every six hours an ounce of almond oil containing 1 per cent of iodoform

(1) Revue Neurologique, Dec 30, 1904.
(2) Jour. Am. Med. Assn., April 1, 1905.
(3) Neurolog. Centralblatt, May 16, 1905.
(4) La Clinica Moderna, Sept. 21, 1904.

was injected. [The case is doubtful, however.—ED.] Jirasek[1] reports two seizures of tuberculous meningitis in the same individual as cured, the two attacks being separated by an interval of two years. Kummell[2] has operated upon several cases of tuberculous meningitis, but not one recovered.

A. Napier and J. Anderson[3] report an interesting case of tuberculous meningitis; at first thought to be typhoid fever. [The persistent vomiting occurring early in the case is not suggestive of typhoid fever to the Editor, nor is the retention of urine which necessitated catheterization, and which is mentioned as a symptom in the case.] The gurgling in the right iliac fossa, the pain on pressure, the abdominal distension, the spots, and the dry, brown and fissured tongue seem responsible for the diagnosis made, notwithstanding the fact that Widal's test twice gave a negative result. Post mortem examination disclosed a tuberculous cerebrospinal meningitis.

More puzzling is the case of Cheney[4] which is reported as one of primary tuberculous meningitis. The patient who was about 3½ years of age had never been previously sick, and when taken ill, was the picture of health. The first symptom was vomiting, attributed to a gastroenteritis from popcorn. At this time the pulse and temperature were normal. Subsequently the child showed increasing lassitude, but the reflexes and pupils were normal. By the seventh day the vomiting was persistent. During the second week the vomiting and inactivity of the child continued. There was no change in reflexes or pupils, no flatness of the abdomen, retraction of the head or neck, muscular twitchings or convulsive movements. On the fourteenth day the drowsiness increased almost to coma, and at times the pupils were unequal. Subsequently pronounced meningitic symptoms appeared, and tubercle bacilli were found in the cerebrospinal fluid obtained at the third puncture. A post mortem examination proved the correctness of the diagnosis.

Syphilitic Meningitis. Schaffer[5] reports a case of ex-

(1) Revue Neurologique, Feb. 15, 1905.
(2) Neurolog. Centralblatt, May 16, 1905.
(3) Glasgow Med. Jour., June, 1905.
(4) Jour. Am. Med. Assn., July 8, 1905.
(5) Neurolog. Centralblatt, Nov. 16, 1904.

tensive syphilitic meningitis involving both the convexity of the brain and the base, in a woman 44 years of age, and single. The Jacksonian epilepsy, present as a symptom, found its anatomic origin in the changes over the motor zone, and since these changes chiefly affected the anterior portion of the middle convolution, the beginning of the attack in the left upper extremity is explained. Besides the Jacksonian epilepsy, the patient suffered from attacks of apparently genuine epilepsy, with amnesia, biting of the tongue, and incontinence. There was also a left hemiparesis due to partial destruction of the right motor zone. Complete tactile anesthesia was present in the fingers, gradually fading out as the trunk was approached. The left hand showed stereoagnosis, or, more properly, was stereoanesthetic. Other interesting features were obstinate headache, somnolence and impulsive laughter.

Drouet[1] reviews the subject of acute syphilitic meningitis now readily diagnosticated by lumbar puncture and cytodiagnosis. It seems much more frequent than was supposed. Before 1900 only 2 incontrovertible cases were on record; since then there have been 6 others. The symptoms are headache, vertigo, vomiting, neurasthenic disturbances and fever, which may be high; later there is restlessness or somnolence, epileptiform convulsions or coma, and notably ocular and facial paralyses. There may be ameliorations or exacerbations in the course. The duration is usually 26 to 35 days, and the usual termination is in recovery. This form of meningitis is therefore less dangerous than other forms, and is even more benign than most specific complications. It is best treated by antisyphilitic measures. The diagnosis is based wholly on the cerebrospinal fluid and the history of a chancre.

Ballet and Rose[2] report a case which symptomatically showed merely a mental confusion such as is frequently met with in the course of intoxications or the puerperal state, post mortem examination of which showed syphilitic sclero-gummatous meningitis of the right frontal lobe. There was persistent right frontal headache. Death took place in an epileptiform convulsion.

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(1) Revue Neurologique, Jan. 30, 1905.
(2) Ibid., Feb. 28, 1905.

Otic Meningitis. Ignaz Hofer[1] classifies the meningitic complications springing from middle ear disease as follows: (1) Inflammations of the cerebral membranes: (a) Pachymeningitis externa circumscripta (extradural abscess); (b) pachymeningitis interna circumscripta (intradural abscess, also called subdural or cortical abscess); (c) leptomeningitis diffusa; (d) meningitis serosa. Cases are reported in illustration.

Diffuse sarcomatosis of the arachnopia of brain and cord with characteristic changes in the cerebrospinal fluid, is reported at length by W. Rindfleisch.[2] Three cases are reported, all offering great difficulty in a differential diagnosis between meningitis and sarcomatosis. Autopsies were performed in only 2 of the cases. Microscopically there were vast numbers of tiny foci scattered by the lymph-channels. The cerebrospinal fluid was increased, and coagulated spontaneously since it contained a very large amount of albumin. The cells in the fluid were much increased in numbers. In 2 of the cases the fluid was of a faint yellow color, and in the other a dark brownish yellow. The article is accompanied by two plates and a careful bibliography.

Stanley Barnes[3] also reports 2 cases of diffuse sarcomatous infiltration of the spinal pia, with post mortem findings. In addition to his two cases, nine others from the literature are reported, which with the three of Rindfleisch's makes fourteen in all. Barnes concludes as follows: "(1) If a growth originates in the caudate nucleus or in any other part of the brain and eventually finds its way to the lining of the ventricular cavities, it may 'infect' the cerebrospinal fluid. Metastatic growths may then occur in the following situations: (1) In the lining walls of the lateral, third or fourth ventricles; (2) in the meninges (subarachnoid) at the base of the brain; (3) in the pia mater and arachnoid around the spinal cord; and (4) in the posterior root ganglia, particularly of the cauda equina. (2) In all probability diffuse sarcomatosis infiltration of the spinal pia arachnoid, which is occasionally found post mortem, as in all cases the expres-

(1) Wiener med. Wochenschrift, Jan. 28, 1903.
(2) Deutsche Archiv für Nervenheilkunde, XXVI, p. 135.
(3) Brain, Spring, 1905, p. 30.

sion of a sarcomatosis infection of the cerebrospinal fluid as a result of some primary growth which lies exposed to the stream of the cerebrospinal fluid high up in the nervous system. (3) The regions occupied by such secondary growths are exactly the same as those inflamed by tubercular meningitis after the rupture of a tubercular 'tumor' of the brain." A bibliography follows.

Meningeal Tumor. Two cases of meningeal tumor treated by ligature of their vessels are reported by C. C. Brush,[1] one in an 18-year-old patient sick only six weeks, and the other in a 16-year-old patient who had suffered for seven years. In the first case the tumor was situated over the right motor cortex, but was not removed. Instead its entire blood supply was cut off and the wound closed. In the course of the next year the general symptoms all disappeared, but the motor disturbances of the left arm persisted. In the second case the tumor was found in the region of the right leg center and was treated in the same way with the same result. Brush concludes that such tumors need not be removed, the cutting off of their blood supply being sufficient.

Cyst of the Spinal Dura Mater. A case is reported by A. Schmidt of Dresden,[1] which gave the symptoms of an extramedullary tumor, and which was successfully removed. The case was that of a 16-year-old lad, who had complained of pain in his back for a year and a half. There was some fever, difficulty in micturition and defecation, stiffness of the legs and cramp-like contractures. The spinal column was not deformed, or sensitive to jars or blows. The Babinski and anterior tibial sign were both present. Ultimately a topical diagnosis was made of a tumor in the 5th to 7th dorsal segments; instead, a cyst of the dura was found and removed. Recovery slowly followed.

Dupré and Devaux[2] presented at the April meeting of the Société de Neurologie of Paris a case of double cerebral abscess with diffuse necrotic lesions of the cortex in a tuberculous patient, the symptoms being those usually referrable to a subacute meningitis. Similarly, Achard and

(1) Jour. of Nervous and Mental Diseases, May, 1904.
(2) Deutsche Archiv für Nervenheilkunde, XXVI, p. 318.
(3) Revue Neurologique, April 30, 1905.

Louis Ramond[1] presented at the November meeting a case of cerebromeningeal hemorrhage where the symptoms were those of meningitis. The stiffness of the neck, Kernig's sign, the curled-up posture in bed, the vomiting, the febrile temperature all favored the latter diagnosis, and the amber color of the cerebrospinal fluid, together with the numerous lymphocytes suggested its tuberculous nature. Post mortem examination, on the contrary, showed a bloody inundation of the meninges from a ruptured vessel. The lymphocytosis of the spinal fluid is to be looked upon as entirely due to the irritating effect upon the meninges of the extravasated blood.

Typhoid Meningism. Carl Straubli[2] calls attention to the meningitic symptoms frequently met with in typhoid fever with post mortem findings, in an extensive and very thorough article of 27 pages, and he reports at length 3 cases of his own. The first of these showed no meningitis. Unfortunately, no bacterial examination of the meninges could be made. The second case recovered and was a typical case of typhoid meningism. The third case, with symptoms of meningism or meningitis, resulted fatally, and post mortem pathologic and bacterial examination showed the typhoid bacilli, the case therefore being meningo-typhoid, or typhoidal meningitis. In the latter case a purulent cerebrospinal meningitis was certainly caused by the typhoid bacilli.

HEMIPLEGIA.

Etiology. H. Schmidt[3] was impelled to investigate the possible bearing of the condition of the weather upon the frequency of apoplexy by a chance remark of von Leyden that cases seemed more numerous in spring and autumn than at other times of the year. An examination into the histories of 55 patients from von Leyden's clinic demonstrated no relation between the barometric pressure and the incidence of apoplexy. An investigation was made of 4,413 fatal cases of apoplexy in Berlin from 1899 to 1903

(1) Revue Neurologique, Nov. 30, 1904.
(2) Deutsche Archiv für klin. Med., Dec. 12, 1904.
(3) Wiener klin.-ther. Wochenschrift, Dec. 11, 1904.

inclusive, and it seemed evident that great barometric fluctuation caused hemiplegics to die, but that the barometer and the incidence of apoplexy are not in any way etiologically connected. James W. Russell[1] has investigated precisely the same subject, stating that R. H. Walter[2] had suggested that in certain cases a high barometric pressure is a factor in deciding the onset of an attack of cerebral hemorrhage, especially when a sudden rise of the barometer follows a more or less continued period of depression. Russell's cases of cerebral hemorrhage investigated were those at the Birmingham General Hospital from 1891 to the end of 1902, 66 in number, all verified by post mortem examination, and they indicated that the greater number of cerebral cases occur with marked changes of barometric pressure. As to seasons, more cases occur in the colder than in the warmer months of the year. It was found, too, that a sudden rise of temperature on any particular day is slightly favorable to an attack.

Symptomatology. Pierre Marie[3] at the November meeting of the Société de Neurologie of Paris called attention to bilateral *myosismia* (muscular jerkings) observed in the lower extremities in fresh organic hemiplegias. Up to this time no one has called attention to this symptom, which consists of a sort of fibrillary tremor or, better, a fascicular tremor, sweeping over the muscles in broad waves, yet involving only a small portion of a muscle at a time. These waves seem slower, less brusque, than those of ordinary tremor or of myoclonus. Hence, Marie's invention of the term *myosismia* (earthquake-shocks in the muscle). The seat of such movements is especially apt to be the anterior thigh muscles, the quadriceps femora; but they also exist in the calf, and sometimes along the inner edge of the foot. Marie has never seen them in the upper extremities. Curiously enough, *myosismia* occurs not merely on the hemiplegic but also on the sound side, and on both sides with about the same intensity. Marie has observed it in softening (encephalomalacia) as well as in hemorrhage. Indeed, it seems to occur chiefly in fresh cases for the most part. The intensity of the hemi-

(1) Lancet, Jan. 28, 1905.
(2) British Medical Journal, April 9, 1904.
(3) Revue Neurologique, Nov. 30, 1904.

plegia has no effect upon it. It is probably caused by degeneration of the white substance, secondary to the lesion. Its bilateral distribution is due either to the fact that in hemiplegia the degeneration of the white substance is bilateral or because of bilateral innervation from one hemisphere, well known in the case of the lower extremities. Marie affirms that *myosismia* never occurs in hysterical hemiplegia, and is, therefore, a differential sign.

Homolateral Hemiplegia. Dupré and Camus[1] report a case of left homolateral hemiplegia in a weak left-handed person who showed an old right infantile hemiplegia, the cause of his being left-handed. Cases of true homolateral hemiplegia due to congenital absence of pyramidal tract decussation are excessively rare, cases having been published by Bidon and Zenner. Cases of symptomatic homolateral hemiplegia are more common, the decussation existing, but the hemiplegia being due to a meningeal collection compressing the opposite peduncle, or some other similar causes. Dupré and Camus regard their case as a true one, due to congenital absence of the pyramidal decussation. The clinical picture was a common one: bronchopneumonia, acute vegetative endocarditis, cardiac thrombosis, cerebral and renal embolism, with softening of the left frontal lobe secondary to thrombosis of the left middle cerebral artery. The infantile paralysis had evidently led to an agenesis of the pyramidal system, the right anterolateral column in the cord being very much more slender than the left. Moreover, the decussation of the pyramids was found, incomplete and unsymmetric, the right pyramid being smaller than the left.

Pringle[2] also reports a case of homolateral, or, as he calls it, "collateral," hemiplegia due to a cerebral trauma, there being a fracture of the skull, the fissure commencing anterior to the right parietal eminence, and running downwards and forwards, bifurcating into two limbs. There was found post mortem extensive laceration of the right frontal and temporo-sphenoidal lobes, with a large effusion of blood in the right middle and in the right anterior fossæ. The hemiplegia was in the right side. Noth-

(1) Revue Neurologique, March 30, 1905.
(2) Scottish Med. and Surg. Journal, November, 1904.

ing is stated by the author concerning the post mortem condition of the decussation of the pyramids.

Conjugate Deviation of Head and Eyes. Brissaud and Pechin[1] object to the term of "conjugate deviation of the head and eyes" as too exclusive, though it deserves an important place in nosology. Sometimes the eyes alone deviate, sometimes both eyes and head, sometimes only the head deviates. Recent investigations show that the absolute muscular power of each of the ocular muscles is diminished on the two sides, but especially on the hemiplegic one; and that the rotation of the head may be due to unilateral sensory anesthesia. What Brissaud insists upon, however, is that there is properly no deviation of the eyes, but an ocular hemiplegia, a hemiophthalmoplegia, just as there is a general muscular hemiplegia. Naturally both eyes are affected by a one-sided lesion because of the synergetic action of the two sets of ocular muscles, exactly as a unilateral occipital lesion produces a hemianopia. The term "ocular hemiplegia" best expresses this view of conjugate deviation. The turning of the eyes is not spastic at all. The eyes are turned because in such a position they are at rest or in dynamic equilibrium. An illustrative case from Brissaud's service is reported. It will be noted that in all hemiplegics with such deviations the eyes cannot be brought past the mid-line.

Grasset[2] reports a case of conjugate deviation of the eyes with hemianopia, in a man 62 years of age, showing also a left hemiplegia and left hemianesthesia. The eyes were constantly turned to the right, it being impossible for him to turn them past the midline toward the left. A large hemorrhage was found occupying the right optic thalamus and all the adjacent portion of the internal capsule. It had not ruptured into the ventricle. The main feature is that the hemiplegia is left, while the deviation of the eyes is to the right. The patient, therefore, looked toward the side where he could see. Grasset questions whether this is coincidence or whether it expresses a definite relationship. Cannot the deviation be sensory in origin? What can be the part of hemianopsia in conjugate deviation? For Grasset each hemisphere sees and looks with both

(1) Progrès Médical, Aug. 13, 1904.
(2) Revue Neurologique, July 15, 1904.

eyes towards the opposite side, there being an actual relation for him between the location of the vision-centers for the two eyes and the ways the eyes turn. A lengthy discussion of the symptom is given. A very thorough exposition of Bard's view of the symptom appears in *La Semaine Medicale,* Jan. 13, 1904.

Déjerine and Roussy[1] report a case of hemiplegia with conjugate deviation of the head and eyes in a congenitally blind woman, 71 years of age. This case apparently wholly invalidates the theory of Bard and that of Grasset, who maintains the existence of a posterior bilateral cortical oculomotor center situated in the gyrus angularis. The conclusions of Déjerine and Roussy follow: "(1) If hemianopsia and conjugate deviation of the eyes are symptoms frequently associated in the same subject (Joanny Roux, Bard, Grasset, Dufour), hemianopsia is not necessarily the cause of the deviation, since our case demonstrates that such conjugate deviation of the head and eyes may exist in the congenitally blind; subjects, therefore, in whom there can be no cortical visual area, it never having been educated. (2) Conjugate deviation and rotation of the head are not invariably, as has been said, of paralytic origin when the patient looks toward the side of the lesion. This is proved by the fact that correction of the deviation is possible (first case of Dufour), and by the state of contraction of the cervical muscles on the sound side of the neck in our case. (3) The cortical center of innervation for the movements of the head and eyes is by no means unique in its localization or in its projection-fibers, because, as Grasset and we have had occasion to observe, there may be a dissociation of the syndrome, rotation of the head to one side and conjugate deviation of the eyes to the opposite side."

Dissociation of Sensation. Schaffer[2] reports a case of cerebral hemianesthesia in which there was topoanesthesia (inability correctly to localize the points touched by a pin-point), and dissociation of sensation on the hemianesthetic side. Warmth was almost without exception unperceived, and cold was perceived almost without exception, and regarded as painful. Sensibility of the right half of the

(1) Revue Neurologique, Feb. 15, 1905.
(2) Neurologisches Centralblatt, June 16, 1905.

tongue and of the palate is perfect In a second paper[1] he discusses the subject at greater length, and an important discussion follows.

Liepmann[2] reports in some cases of cerebral hemiplegia a dissociation of the pain-sense in that the superficial pain-sense may be preserved and the deep lost. [The case itself is convincing but it is not a proper one from which to draw general conclusions.—ED.]

Increase of the Cutaneous Reflexes on the Paretic Side in Organic Hemiparesis. Redlich[3] reports 7 cases in whom organic cerebral lesions existed, one being confirmed by autopsy. In all of these instead of finding the skin-reflexes absent or reduced, according to rule, on the hemiparetic or hemiplegic side, they were present or even more pronounced than on the sound side; or else in a given case some only were reduced, all the other reflexes being increased.

Amyotrophy. Weisenburg[4] affirms that atrophy was present in every one of his 160 cases studied. The atrophy probably first manifests itself in the small muscles of the hand and in the muscles of the shoulder, being more marked in those cases in which the paralysis is more marked, and greater in the digital than proximal end of an extremity. The lower limb is never so much affected as the upper limb.

Caracciolo[5] in some experiments on cortical ablation on dogs found that the trophic disturbances following in the muscles were due to the spread of the degeneration from the brain down the pyramidal tracts to the anterior horn cells. Next followed changes in the motor cells of the anterior horn and muscular atrophy.

Temperature on the Hemiplegic Side. It is usually asserted following the researches of Luys, Féré and Strümpell, that the temperature of the paralyzed side is lower than that of the sound side. Raymond and Courtellemont, in the April meeting of the Société de Neurologie of Paris, showed a case in whom the hemiplegia had been in existence three months, and yet the temperature of the

(1) Neurologisches Centralblatt, June 16, 1905.
(2) Ibid., Aug. 16, 1904.
(3) Ibid., May 1, 1905.
(4) Jour. Am. Med. Assn., Feb. 25, 1905.
(5) Rivista sper. de Fren. XXX, September, 1904.

paralyzed side was higher than that of the sound side. Parhon and Papinian, in the March, 1905, meeting,[1] showed a similar case where after several months the rule was still reversed. In this latter case the site of the lesion was thought to be the optic thalamus.

Pre- and Post-Hemiplegic Pain. Weisenburg[2] calls attention to the occurrence of pre-hemiplegic pain, manifested chiefly in the muscles and joints of the upper extremity. Sometimes they are present in the chest and side of the face. In some instances the pain ceases with the onset of the hemiplegia. If the hemiplegia is due to a thrombosis the pain is easily explained. Post-hemiplegic pain is very common, only 5 in a series of 109 cases being without it. The parts involved are chiefly the various joints and muscles of the paralyzed side. Other patients complain of painful paresthesias.

Treatment. Foerster[3] remarks that after any hemiplegia a tendency to spontaneous restitution is observable, but in this tendency not all the muscles share equally. Some groups, indeed, tend to remain permanently paralyzed. In the leg the dorsal flexors of the foot are backward; and in the thigh, the quadriceps group. In the arm it is the extensors which are specially weak. Hence, treatment by muscular exercises is very important as a training for these groups. The first thing is to teach the forgotten movement-complexes anew. For this passive motion by the physician may aid the patient's efforts. Then such complex movements as standing, walking, sitting, walking up stairs, running, etc., may be attempted after simple movement-complexes are acquired. Probably the subcortical centers are awakened to activity by this means. Meige[4] advocates these same exercises, declaring that in hemiplegia the chief effect is motor amnesia, the memory of putting into effect complex movements being lost. There is really a motor abulia. Exercise may alone restore to an hemiplegia the ability to rise, sit down, go up or down stairways, eat, write, etc. Meige declares that in many instances he has secured very happy results.

(1) Revue Neurologique, March 30, 1905.
(2) Jour. Am. Med. Asso., Feb. 25, 1905.
(3) Neurolog. Centralblatt, Nov. 1, 1904.
(4) Revue Neurologique, Feb. 15, 1905.

Cerebral Hemorrhage. In 1903 Crouzon called attention to subpial hemorrhage occurring secondary to primary cerebral hemorrhage. Thus it happens that the cerebrospinal fluid may be rose-tinted and show the presence of blood without necessarily implying that the primary intracerebral hemorrhage has broken through the cortex to the subarachnoid space. Fage and Faure-Beaulieu[1] report a similar case. The secondary subarachnoidean hemorrhage seems to be due to the cerebral and medullary commotion consequent upon the primary hemorrhage, this of itself acting as a traumatic agent.

Froin and Laderich,[2] on the other hand, report a case of cerebral hemorrhage with inundation of the ventricles and subarachnoid spaces, in which lumbar puncture gave varying results. The cerebrospinal fluid was colored with hemoglobin; moreover, the color varied from time to time in the same puncture, due not to the varying numbers of red corpuscles which it contained, as might be thought, but to a real variation in the amount of contained hemoglobin.

Lacunar Cerebrosclerosis. In a former volume of the Practical Medicine Series attention was called to Marie's lacunar softening findings in the brains of old hemiplegics. J. Grasset[3] discusses the matter in great detail under the caption cerebrosclerosis. Although the word signifies the opposite of the word cerebromalacia, Grasset declares it to be the old French "softening," dating back to 1820, when it was first described by Rostan. Clinically the hemiplegia is very slowly progressive. The patient walks with very short steps, and on this account old observers looked upon it as a kind of cerebral softening. In reality it is a cerebral sclerosis, progressive in type, and originating in the arteries. The subjects are victims of arteriosclerosis. Often hemorrhage and true softening occur as complications and confuse the pathologic picture. The arterial lesion produces a lacunar dystrophy, at first an atrophy of nerve elements, then their disappearance with the formation of a lacuna, which forms a cyst or a cicatricial sclerosis. They are habitually found in the basal ganglia. Grasset distinguishes seven types: (1)

(1) Revue Neurologique, March 30, 1905.
(2) Gazette des Hôpitaux, Feb. 14, 1905.
(3) La Semaine Med., Oct. 19, 1904.

Cerebrosclerosis with gradual onset and slow development; (2) a form reminding one of pseudo-bulbar paralysis; (3) a pseudo-paralytic form, like general paralysis, with remissions and retrogressions; (4) cerebrosclerosis of ordinary hemiplegia; (5) the transitory and recurrent hemiplegias of uremics; (6) epilepsy coming on in an advanced age; (7) a rare form, beginning with medullary symptoms, of the labio-glosso-laryngeal type.

"Worm-eaten" Appearance of the Cerebral Cortex. Dougherty,[1] studying in Marie's laboratory, describes a peculiar appearance, consisting of a superficial excavation of the cortex of the brain, generally of a brownish-yellow color, found principally on the *basis cerebri* and rarely on the cerebellum. It is found only in old age and is not very common. Of 180 brains recently examined, only 4 showed the condition, about 2 per cent. One of the 4 cases suffered from senile epilepsy. All of them presented a marked diminution of the mental faculties. The pathogenesis of the lesion is not yet established.

INFANTILE CEREBRAL HEMIPLEGIA AND INFANTILE, ETC., DIPLEGIA.

Hoffmann[2] reports cases of infantile cerebral hemiplegia in a brother and sister, the etiology being unsual. Each case began as an acute meningitis, one being followed by a cerebral form of paraylsis with athetosis, ankle-clonus, and Babinski's sign, the other being followed by a monoplegia of spinal origin with muscular atrophy. Probably there was gastrointestinal infection, followed by a cerebrospinal meningitis which in one occasioned a polioencephalitis and in the other caused a poliomyelitis.

Faure-Beaulieu and Lewandowsky[3] report an interesting case of infantile cerebral paralysis in a man of 26 years in whom athetoid movements were continually present, and whose muscles on the paralyzed side were hypertrophied, due to the spasticity present.

(1) Revue Neurologique, Dec. 30, 1904.
(2) Ibid., May 15, 1905.
(3) Ibid., Feb. 28, 1905.

Sicard[1] reported a similar case in the January meeting of the Société de Paris. The patient, it turned out in the discussion, had been in Brissaud's service in 1893, his case having been reported at the time, when he showed a double athetosis without hypertrophy. At present the hypertrophy of the right upper limb is relatively enormous. As an illustration of muscular hypertrophy of cerebral origin the case is an excellent one.

Little's syndrome, says Paul-Ernest Carrel,[2] is either congenital or is acquired in the first months of life. It is difficult to classify it into types according to etiology or symptomatology. There is, furthermore, no anatomic lesion peculiar to it, and the pathogenesis is various. Hence, it is properly a syndrome and not a disease.

Spiller[3] discusses Little's disease under the title: "Congenital Spastic Rigidity of the Limbs or Congenital Hypertonia," and reports 2 cases in addition to the 2 he reported in 1898 in the *Journal of Nervous and Mental Diseases* (p. 81.) In one of the later cases there was a failure of development of the pyramidal tracts in the spinal cord, yet without macroscopic lesions. In the other, Little's syndrome seemed caused by pressure of the cervical vertebræ upon the spinal cord, this giving rise to the so-called "spinal type" of Little's disease, reported by Déjerine in 1903, and mentioned in the Practical Medicine Series. In each case hypertonia was pronounced.

Lewandowsky[4] reports a case of chronic hemichorea due to an infantile cerebral lesion with an abnormal reaction to faradism in the muscles. Instead of tonus being obtained there were a series of clonic contractions brought forth by the faradic current, there being 120 to 200 per minute. With galvanism there was merely a quantitative reduction in the muscular reaction.

Interesting in connection with athetosis in infantile cerebral hemiplegia and diplegia is a case reported by Brissaud and Sicard.[5] There was absolutely no evidence of any paralysis or of any lesions of the central nervous system, except a marked and double athetosis. The in-

(1) Revue Neurologique, Jan. 30, 1905.
(2) Ibid., May 30, 1905.
(3) Univ. of Penn. Med. Bulletin, January, 1905.
(4) Revue Neurologique, Feb. 28, 1905.
(5) Ibid.

voluntary movements were so active that the patient could not muster sufficient mechanical skill to make her living at any trade. Evidently, marked athetosis may occur, as in this case, without hemiplegia necessarily being present.

Treatment. Spiller and Frazier[1] discuss the treatment of cerebral palsies and athetosis, and advise nerve anastomosis and transplantation. If the hemiplegia is complete, or nearly so, little can be done; if, however, it be incomplete there is hope. For example, in cases where the flexors alone recover power, the central ends of some of the least important flexor nerves may be joined to the peripheral ends of the extensor nerves, and in this way the normal balance between flexors and extensors may be secured. If the flexors of the toes be stronger than the extensors, an anastomosis between the central ends of one or more flexor branches and the distal ends of the severed extensor nerves, is indicated. Even though hemiparesis may have existed many years, regeneration of the united nerve-ends will occur, and function follow, provided the muscles involved be not entirely wasted. For curing athetosis Spiller proposes that the motor nerves supplying the muscles concerned be cut and immediately sutured again; in this manner the involuntary activity would be lessened. Or flexor nerves might be joined to extensor muscles or *vice versa*. A case so operated upon by Frazier was distinctly improved. Frazier also recounts the case of a hemiplegic woman of 65 years, who showed much greater paralysis in the extensor than in the flexor group in the upper extremity, and who was distinctly helped by implanting one-half of the median nerve into the musculospiral.

CEREBRAL TUMORS.

Symptomatology. Franceschi[2] records the case of a girl whose first symptom was a continual need for sleep. The classical symptoms of tumor, headache, vertigo, vomiting, dimness of vision, did not appear until after the somnolent tendency. The sleep was calm and natural; the patient could be aroused easily, but she went to sleep while being

(1) Journal of Nervous and Mental Diseases, May, 1905.
(2) Rivista di Patologia nerv. e ment., October, 1904.

spoken to. There was no diminution of the mental faculty. At the autopsy a small sarcoma was found in the interpeduncular space, behind the chiasma, but partly involving it. The somnolence seems best explained on the basis of cerebral amnesia due to compression of the basilar artery, exerted by the tumor.

Auditory Disturbances. Souques[1] calls attention to the various reasons for deafness, total or partial, in cerebral tumors. Most cases are unilateral, and are due to a direct lesion of the auditory pathways. Yet in some instances when the latter are intact, disturbances of hearing still occur. These cases are apt to be bilaterally deaf. An interesting case is recounted of a man of 40 years, who showed normal intelligence, and bilateral deafness as well as complete blindness. There was some hyperesthesia in the territory of the left trigeminal nerve, a facial tic on the same side, and deafness. This suggested a basal tumor. At the autopsy the tumor was found in the left prefrontal lobe. The auditory pathways were not harmed and the middle ears were normal. Unfortunately, the internal ears were not examined. Before the signs of tumor, however, there was no trouble with hearing. Souques believes the deafness to have been due to an increase in the intracranial pressure. In general, he affirms, tumors which develop rapidly, may cause alterations in the internal ear by increasing the pressure or hypertension of the cerebrospinal fluid, and this may lead to a sort of labyrinthitis or cellulitis of Corti, analogous to the choked disc in optic neuritis. Babinski, in discussing the subject, stated that certain experiments which he had made had led him to conclude that the pressure of the labyrinthine fluid was closely connected with that of the cerebrospinal fluid, and that he, therefore, used lumbar puncture in these case as a remedial agent. Sicard stated that in Raymond's service 4 cases of anosmia in cerebral tumor had been lately observed. These cases supported Souques' explanation.

Cerebellar symptoms occurring in a case of tumor of the centrum ovale are reported by W. L. Ascherson.[2] A single woman, 42 years of age, first noticed a dragging of her right foot, and subsequently became unable to stand or

(1) Revue Neurologique, July 30, 1904.
(2) Lancet, Sept. 10, 1904.

walk. Ultimately the left leg became even more weak than the right, her eyesight failed and she was assailed by excessive headache and persistent vomiting. Before she took to her bed the disturbance of equilibrium and gait was very marked; when she stood without support she tended constantly to fall backwards and to the right. The gait was described as cerebellar. [The author does not report that the cerebellar triad of symptoms, asthenia, atonia, and astasia, were present in a typical cerebellar form.—ED.] A diagnosis was made of a right-sided tumor of the cerebellum, based on: (1) The presence of marked ataxia of the cerebellar type (titubation) with tendency to fall backwards and to the right; (2) the presence of tenderness in the right occipital region; (3) the intensity and the early onset of the optic neuritis; (4) the presence of static ataxia of the right upper extremity; and (5) the presence of nystagmus when the eyes were directed to the right. Operation was advised, therefore, and performed but the patient died soon after from shock. At the autopsy a large spindle-celled sarcoma was found beneath the upper part of the left Rolandic area.

James Collier[1] discusses the false localizing signs of intracranial tumor, his conclusions being based upon 161 cases. The objects of Collier's paper are to show: (1) That local signs appearing late in the course of intracranial tumor, where general signs alone have pre-existed, are often of false portent; (2) that the relative frequency with which local signs have occurred has been due to associated vascular lesions—meningitis, hydrocephalus, local spreading edema of the brain, and metastases; (3) that the absence of usually accepted local signs during the early days of illness in intracranial tumor is in itself a most important localizing indication, confining the disease to the supratentorial region; (4) that true localizing signs at one time present may later become concealed or undemonstrable owing to the development of other signs; and (5) that in cases which come under observation for the first time later in the disease, diagnosis may be difficult, erroneous, or impossible. Each of these postulates is well supported by typical clinical histories. [A case

(1) Brain, Winter, 1904, pp. 490-508.

like Ascherson's is instructive.—ED.] A man of 20 years, with headache, vomiting and optic neuritis, showed paralysis of the left external rectus, marked nystagmus with the slow movement to the left, complete nerve deafness of the left ear, left peripheral facial paralysis, left cerebellar position of the head (head inclined to the left shoulder, and face rotated to the right), marked head-retraction during the paroxysms of pain, bilateral ataxia greater on the left side, with lurching to the left on attempting to walk. The symptoms apparently pointed conclusively to a growth in the left posterior fossa invading the cerebellum and the 6th, 7th and 8th cranial nerves. The autopsy revealed a glioma of the left pre-frontal region. In this case all of the late symptoms were the result of pressure, the 8th nerve showing degeneration, and the left lateral cerebellar hemisphere being much indented by the edge of the foramen magnum. A similar case is reported in a child of 8 years. Paralysis frequently of the cranial nerves exists as a secondary or indirect symptom. Again, Jacksonian epilepsy, hemiepilepsy, and general convulsions may occur, not merely in cortical lesions, but in connection with tumors of the brain-stem and of the cerebellum. The complications due to meningitis, vascular changes and other secondary effects are analyzed. Many baffling symptoms may be due to degeneration of the posterior columns of the spinal cord.

Clark[1] reports the case of a woman, 36 years of age, with signs of brain tumor, but without localizing signs except possibly persistent pain over the left parietal region. A radiograph [accommodatingly] showed a light shadow approximately in the region of pain and tenderness, and trephining was done without finding a tumor. At the post mortem examination the tumor was found in the right superior parietal gyrus. The absence of stereognostic symptoms is commented upon.

Choked Disc or Papillary Stasis. Flatau[2] reports a case of brain tumor in which there was a gradual regression of the papillary stasis, the change being attributed to lumbar puncture which was done nine times. A careful review of the literature and an extensive bibliography follows.

(1) Journal of Nervous and Mental Diseases, August, 1904.
(2) Muench. med. Wochenschrift, April 4, 1905

Numerous interesting clinical cases of variously located cerebral tumors have been reported during the past year. McCay and Thurston[1] report a case of tumor of the right caudate nucleus and frontal lobe without any localizing signs whatever. W. C. Kendig[2] reports a case of brain tumor with post mortem examination, showing merely the occurrence resembling dementia and progressive blindness, running a very protracted course. A very interesting discussion of this and similar cases by Diller, Mills, Keen and others, with special reference to surgical treatment and methods is to be found in the *Journal of the American Medical Association,* March 18, 1905.

Graeffner[3] reports 5 cases of tumors of the central nervous system, 3 of which were cerebral tumors, 2 being cholesteatomata. Marie and Catola[4] report an interesting case of a perithelioma which had infiltrated the white matter of both cerebral hemispheres, and which was invisible macroscopically, though quickly appearing when the brain was put in Mueller's fluid, due to the difference in color between the infiltrated and non-infiltrated areas. Philip Zenner[5] reports a case of tumor of the occipital lobe with an interesting clinical history. Walton[6] and Brownrigg[7] each report cases of brain tumor, each with a post mortem examination. Robb[8] reports the case of a man showing multiple cerebral gummata of large size who showed no symptoms until three days before his death. His condition was then attributed to a scalp wound resulting from a fall. He rapidly passed into coma and died. Liepman[9] reports a case of brain tumor apparently following trauma, which produced a wound over the right parietal bone. At the operation a tumor was removed from the right motor region, but death followed notwithstanding. Oppenheim[10] discusses briefly the symptomatology of tumors in the posterior cerebral fossa. Burns[11] reports 4 good cases of brain

(1) Lancet, April 29, 1905.
(2) Journal Amer. Med. Assn., March 11, 1905.
(3) Berliner klin. Wochenschrift, Dec. 13, 1904.
(4) Revue Neurologique, March 30, 1905.
(5) Journal of Nervous and Mental Diseases, January, 1905.
(6) Ibid., 1905, p. 256.
(7) Ibid., 1905, p. 257.
(8) Ibid., 1905, p. 263.
(9) Berliner klin. Wochenschrift, 1904, No. 36.
(10) Neurolog. Centralblatt, Feb. 1, 1905.
(11) Ibid., June 1, 1905, p. 536.

tumor, in one of which trauma antedated the tumor and in which damages were collected. Behr[1] reports a case showing cerebral metastatic tumors to the number of about 100, the primary tumor being a large one in the region of the right thalamus and especially of the nucleus caudatus.

Fraser[2] reports a case of cerebral tumor occurring in an individual showing a complete transposition of the viscera. Warrington[3] reports a case of tumor of the basal ganglia beginning with loss of memory and hallucinations of smell, which latter turned out to be of absolute localizing value at the autopsy. E. W. Holmes[4] discusses the relation between trauma and cerebral tumor.

Diagnosis. *Cerebral Skiagraphy.* G. E. Pfahler[5] has made a study of cerebral tumors both in the living and upon the cadaver. He finds that a time exposure of one-and-a-half minutes, and a distance of 18 inches gives the best results. The tube used was one with a vacuum of 2 1-2 inches. He believes that a skiagraph will show most large lesions, such as new growths, softening, hemorrhage, and abscess; but the responsibility of an operation upon the brain ought never to be incurred by reason of skiagraphic evidence alone. All patients with suspected cerebral tumor ought, however, to be examined by the x-ray because of the added information thus obtained.

Pseudo-tumors. Nonne[6] in November, 1904, published a paper on cases showing the symptom complex of tumor cerebri, ultimately recovering (pseudo-tumor cerebri). He asserts that "there are types of cases in which our present experience and knowledge justify the diagnosis of brain tumor, but the further course of which shows that the diagnosis was wrong, even though a subsequent critical consideration fails to reveal why the diagnosis was wrong." He then proceeds to detail 8 cases, all of which terminated in recovery. The first case may serve as an example. The patient, a man of 32 years, was under observation for nearly four years. At first he complained of gradually increasing weakness in his right arm, and later in the

(1) Neurolog. Centralblatt, June 1, 1905, p. 542.
(2) Edinburgh Med. Jour., October, 1904.
(3) Liverpool Medico-Chirurgical Journal, 1903-4, p. 227.
(4) American Medicine, Dec. 10, 1904.
(5) American Jour. of the Med. Sciences, December, 1904.
(6) Deutsche Archiv für Nervenheilkunde, XXVII, p. 169.

right leg. Later the right half of the face became paretic. There was no sign of venereal infection. The gait was slightly paretic in the right leg. The cutaneous reflexes were lively on the left side, and weak upon the right. There were no sensory disturbances, and no Romberg's sign. Examination of the fundi, made by an ophthalmologist, showed a bilateral beginning choked disc, the papilla being cloudy and very poorly defined at its margins. Lumbar puncture disclosed a slight increase in the tension of the cerebrospinal fluid. There was headache and nausea. The pulse was 60 to 64 beats per minute. Fever was never present. Rest in bed, a bland diet, and an icebag to the head sufficed to cure the patient, and he is still well. Seven similar cases are reported, in one of which trephining was done, absolutely nothing abnormal being found.

A further series of 4 cases follows with fatal issue, upon 3 of whom autopsies were performed. In these cases the symptom-complex strongly suggests tumor cerebri, yet nothing was found post mortem. Five other cases are reported showing the difficulty there is in differentiating hydrocephalus from tumor cerebri. [The first 2 of these remind one of the cases reported by Byrom Bramwell in *Brain,* where instead of tumor there was found post-mortem a closure of the foramen of Magendie, leading to a simple damming up of cerebrospinal fluid, and so causing a hydrocephalus by stasis.—ED.] Three other instructive cases are added by Nonne, the hydrocephalus being occasioned by injury of the head, psychic trauma, or as a sequel of chronic sinus-thrombosis. Henneberg[1] reports the case of a child who developed Jacksonian epilepsy and symptoms very clearly pointing toward disease of the left motor area. Trephining disclosed normal conditions. Unfortunately, the patient died from subsequent meningitis. Examination post mortem showed no trace of any tumor or local disturbance of the brain. This case is much like the one reported by Oppenheim in 1901 *(Berliner klinische Wochenschrift).*

(1) Neurolog. Centralblatt, April 1, 1905.

ENCEPHALITIS.

Acute Hemorrhagic Encephalitis. E. E. Southard and F. R. Sims[1] report a case of cortical hemorrhages following scarlet fever, the condition developing during convalescence. The basis of the cerebral symptoms was a markedly hemorrhagic encephalitis, focal in the parietal lobes. There were found post mortem, large areas of hemorrhage and a few superficial or meningeal areas of frank suppuration, together with a tremendous increase in phagocytes, crowded into the pial and adventitial meshes, or spaces of lost brain substance which had undergone acute fatty degeneration. Based upon this case E. E. Southard and C. W. Keene[2] made a study of encephalitis in man, due to the staphylococcus pyogenes aureus; and parallel lesions were produced in guinea pigs and simultaneously studied. Six cases are reported from the Boston City Hospital, one being the case secondary to scarlet fever, just mentioned. In all these cases the staphylococcus pyogenes aureus was found. The atria of infection were obscure in 2 cases, but in 3 others acute endocarditis was found, and one was a case of scarlet fever. Focal pulmonary lesions were either grossly prominent or were made out microscopically in all six cases. There was pus in the bronchi of two cases, bronchopneumonia in one case, and septic areas of hemorrhage, septic infarction, and multiple abscesses, in the other three case respectively. [This coincidence of pulmonary and brain lesions emphasizes the old clinical rule. —Ed.] In all cases it was found that the inflammation of the meninges and cortical substance in man produced by the staphylococcus aureus is distinctively hemorrhagic. Very little fibrin is found in these lesions. The bleeding continues in some cases until stopped by intracranial pressure. Small vessels are, as a rule, affected. Experimentally intrapulmonary injections of 0.5 c.c. of a twenty-four hour bouillon culture of the staphylococcus were made in guinea pigs. Exudation into the meninges almost certainly followed. The cell-picture is meningitis, seen in six hours; ependymitis, seen in twelve to fourteen

(1) Journal American Med. Assn., Sept. 17, 1904.
(2) Am. Jour. Med. Sciences, March, 1905.

hours; and exudation into the brain substance, from twenty-four to forty-eight hours.

Raymond and Castan[1] report two cases of acute hemorhagic encephalitis, both occurring in patients with febrile pulmonary affections; and they carefully review the literature. These are among the first cases reported in France. They are not to be set down as cases of purulent infarcts. They seem rather to be due to a phlebitis and venous thrombosis, the arteries remaining unaffected. In the differential diagnosis meningitis is the most difficult to eliminate.

Bullard and Sims[2] report a case of diffuse encephalitis due to the pneumococcus, in a man suffering from severe bronchitis. Suddenly there developed a mild delirium followed by flaccid paralysis of the left side, with rigidity on the right, and incontinence. Death came seven days later. The autopsy showed an extensive diffuse encephalitis, and cultures of the diseased area gave the pneumococcus. The lesions histologically consisted merely of accumulations within and surrounding the adventitia of the vessels of the cortical arterial system. Associated with these cell-accumulations were found punctiform hemorrhagic foci. A review of the literature follows.

Huismans[3] reports a case of *encephalomyelitis hemorrhagica diffusa,* with symptoms closely resembling those of Landry's paralysis. A man, 38 years of age, without any predisposing cause suddenly was seized with generalized nervous symptoms, chiefly headache, and then by weakness in the legs, later becoming paraplegic. Gradually the paralysis ascended, involving the diaphragm, the muscles of swallowing, and finally the respiratory center. Post mortem there was found an encephalomyelitis disseminata, the foci varying from the size of a flea-bite in the left centrum ovale and optic thalamus, to the size of a small pea in the cord. Clinically a differential diagnosis was impossible, and was not made. The chief variation in the symptomatology from Landry's type was the pronounced character of the psychic symptoms present.

Hegler and Helber[4] report a case of acute simple men-

(1) Gazette des Hôpitaux, 1904, p. 829.
(2) Boston Medical and Surgical Journal, Dec. 15, 1904.
(3) Berliner klin. Wochenschrift, Jan. 23, 1905.
(4) Deutsche Archiv f. klin. Med., Dec. 12, 1904.

ingoencephalitis, occurring in a girl 16 years of age, and resulting fatally. Bacterial examination was negative, the cultures remaining sterile, and the tissues showed no bacteria of any sort. Microscopically the brain tissue showed pronounced infiltration about the cortical arteries, the whole perivascular lymphatic space being crowded full of cells, for the most part mononuclear leucocytes. There were no giant-cells found. The etiology of the case was not explained.

SINUS THROMBOSIS.

Spiller and Camp[1] call attention to the infrequency with which cases of autochthonous (primary) sinus thrombosis occur, nearly all reported cases of such thrombosis being secondary. They report in all 3 cases, 2 already having been previously reported, and a new case being added. In all instances spontaneous clotting of blood was due to the general bodily condition. A careful and thorough review of the literature with a good bibliography follows. According to Oppenheim, primary thrombosis is due to marasmus, to exhausting diarrhea in children and to tuberculosis and cancer in adults. It rarely occurs in the course of acute infectious diseases. It also occurs in syphilitic cachexia.

SYPHILIS OF THE BRAIN.

Disseminated Syphilitic Encephalitis. A. M. Barrett[2] reports a case in which the cerebral symptoms appeared soon after infection, at a time when the constitutional symptoms were still marked. He was infected in June. In the latter part of November he had severe headache, only partially controlled by treatment, and soon became forgetful and stupid, so that he was committed to an asylum. On December 18 he died. The pia mater was found to be slightly hazy, and hyperemic at the *basis cranii*. Microscopically there was found a meningitis with

(1) Journal Amer. Med. Assn., Sept. 14, 1904.
(2) Am. Jour. Med. Sciences, March, 1905.

local variations in the severity and character of its elements. The perivascular spaces were deeply infiltrated and the walls of the veins were affected in the adventitia. Very little fibrin was found in the exudate.

According to Herschl[1] the cerebral symptoms occurring in the secondary stage, are as a rule, dependent upon vaso-motor disturbances in the meninges. The cerebral complications of teritiary syphilis may be divided into two groups, the first consisting of purely syphilitic affections such as circumscribed or diffuse gummata and arteritis, either of the basal arteries or those of the convexity, and secondary softening. The second group comprises the meta-parasyphilitic diseases.

CEREBRAL TRAUMA.

Hyperalgesic Zones in the Cervical Region in Bullet Wounds of the Head. Milner[2] reports the case of a young man shot in the right temporal region with a bullet of small calibre. The patient was perfectly conscious, and there were no paralyses. He complained of violent pain extending from the occiput to the vertex, and running down the sides of the neck, both left and right, as far as the clavicles. This whole area was extremely sensitive to touch, the line of demarcation above being along the margin of the lower jaw. Slow voluntary movements of the head caused no pain. Recovery followed from the wound but the cervical pain and the sensitiveness of the occiput and vertex persisted. The hyperalgesic cervical zones correspond to the zones studied by Head of London.

Wilms[3] reports his fifth case of the same nature, in a man, 57 years of age, the wound being in the frontal lobe and the right lateral ventricle. Wilms had explained his earlier cases as due to injuries of the region of the cavernous sinus and consequent destruction of sympathetic fibres. Because of Milner's case and his own last case he is inclined to accept Milner's suggestion that the zones are those discovered by Head.

(1) Wiener klin. Wochenschrift, No. 17, 1904.
(2) Berliner klin. Wochenschrift, No. 17, 1904,
(3) Ibid., No, 36, 1904,

Veraguth[1] reports the case of a man seen five minutes after he was shot through the forehead. He fell backward but did not lose consciousness. "My God! He is dead," cried his wife. "No, Helen," he replied calmly, "I am still alive." He implored her forgiveness and consigned his children to her care. He was very thirsty when the physician arrived. He himself told the latter his telephone number when the doctor asked it of those standing about. He was perfectly lucid and absolutely clear as to time and place, and calmly told every detail of what had occurred. He spoke readily and articulated perfectly. His pupils reacted normally. His left side was completely paralyzed and sensation was lost all over it. His pulse was rapid and small, but his respiration quiet and regular. About twenty to thirty minutes after being shot he began to grow delirious, and subsequently lost consciousness, dying in a little less than twelve hours' time. At the post mortem examination the mesial surface of the right frontal lobe was found destroyed. The bullet then passed through the centrum ovale to the interparietal fissure where it stopped.

Dercum[2] reports the case of a mill-hand who was twice hit with a base ball bat over the head. The interesting symptoms were ataxia and nystagmus, notwithstanding that the lesion was in the foot of the second frontal convolution, as shown by the operation performed by W. W. Keen. Tumors of the frontal lobe may give rise to ataxia, which is sometimes bilateral, as in the present case. The nystagmus which was present calls to mind the fact that the second frontal convolution is a center standing in direct relation to ocular movements, as demonstrated experimentally by Ferrier, Beevor, Horsley, Mott and Sherrington. So far as Dercum knows, nystagmus from injury to this part has not as yet been reported, however, except a case of Klien's in the *Zeitschrift für Nervenheilkunde*, 1904, p. 327. Interesting in Dercum's case, too, is the fact that the convulsions which occurred were general in character and not focal. Whether his conclusion that all injuries of this gyrus causing convulsions will always cause general ones is open to question.

(1) Correspondenz-Blatt für Schweizer Aerzte, 1904, p. 354.
(2) Jour. of Nervous and Mental Diseases, 1905, p. 106.

E. Bloch[1] reports a case of fracture of the base of the skull which resulted in paresis of the right side of the body and of the abducens nerve. So far as Bloch can find, this is the first case of abducens paralysis occurring in fracture of the *basis cranii*. Evidently, the right petron was fractured, causing a blood clot compressing the 6th nerve.

O'Hanlon[2] reports an interesting case of delayed results of a wound of the brain, caused by a pistol ball. The man who was shot was up and about the day after and worked continuously the following months. Subsequently convulsions developed. Eighteen months after being shot he died, and the autopsy revealed the pathway of the bullet through the left temporal lobe to beneath the frontal lobe of the right hemisphere. An abscess had formed, containing a small piece of lead to which adhered a sharp piece of bone.

Hedges[3] reports a case of marked mental improvement following an operation for depressed fracture of the skull in a boy 8 years of age. The accident had taken place four years previously. The moral obliquity and destructiveness shown by the boy were also corrected by the operation.

CEREBELLUM.

Tumors. C. K. Mills[4] enumerates the focal symptoms of cerebellar tumor as "vertigo, ataxia, nystagmus, asthenia, spasticity, convulsions and certain brain symptoms." Ataxia is not always present. If the worm is invaded, incoordination of one sort or another is always present. Nystagmus may be vertical or horizontal. Asthenia, ataxia and atonia are perhaps the cardinal symptoms. Among accidental symptoms are those of the cranial nerves. As to treatment, Mills advises operation unless the patient is threatened with immediate death.

Gordinier[5] reports three cases of cerebellar tumors and discusses their symptomatology and localization. Besides

(1) Neurolog. Centralblatt, 1904, p. 940.
(2) N. Y. Med. Jour. and Phila. Med. Jour., Jan. 21, 1905.
(3) Medical Record, Jan. 28, 1905.
(4) St. Louis Medical Review, Oct. 22, 1904.
(5) Albany Medical Annals, February, 1905.

the usual and very well-known symptoms he calls special attention to a coarse intention tremor resembling closely that of multiple sclerosis. Severe vertigo with marked cerebellar ataxia as a rule indicates a lesion of the worm. If the ataxia occurs early the probabilities are that the lesion originated in the middle lobe. The fact that the patient always staggers toward the same side is of no great clinical value in locating the hemisphere affected, as it is impossible to know whether the lesion is irritative or destructive. Cranial nerve symptoms usually occur first on the side of the lesion, such as internal strabismus, facial paralysis, either motor or sensory, deafness and retraction of the head to one side.

T. Granger Stewart and Gordon Holmes[1] discuss extensively the symptomatology of cerebellar tumors, based upon a study of forty of their own cases, and in an appendix describe twenty-two cases of cerebellar and extracerebellar tumor personally observed, in which the diagnosis was confirmed by operation or by autopsy. Of early symptoms headache is an early and almost constant symptom and is usually occipital. Vomiting is an almost invariable symptom during some stage of the growth. Optic neuritis is in their experience a constant accompaniment and seems to be one of the earliest signs, often out of proportion to the general symptoms, its onset as a rule being acute. Vertigo and auditory symptoms are especially common. Of the motor symptoms hemiparesis on the side of the lesion is noteworthy. The absence of rigidity and the normal state of the superficial reflexes indicate that the hemiparesis is independent of any interference with the pyramidal tracts. Associated with the unilateral weakness is a defect of tone (atonia). While the hypotonia of tabes dorsalis and multiple neuritis is always accompanied by loss of the tendon reflexes, the hypotonia of cerebellar lesion may be associated with definite increase of those reflexes or the latter may be absent or diminished in various degrees. Some degree of ataxia of the homolateral limbs is one of the classical signs of unilateral cerebellar tumor. The authors do not agree that Babinski's symptom of "diadocokinesia" is constantly present in all cases of cerebellar

(1) Brain, Winter, 1904, p. 522.

lesions, and they do not regard it as pathognomonic of cerebellar disease. The attitude is considered important, the head being slightly bent toward the side of the lesion and so rotated that the chin is directed towards the shoulder opposite the lesion and the occiput toward the side of the lesion. This so-called cerebellar attitude (an illustration of which occurs in the Practical Medicine Series, September, 1904) also occurs in pontine and mid-brain tumors, and (rarely) in frontal neoplasms as well. In some cases there may be a tendency to retraction of the head. Involuntary movements, such as tremors, occur.

In cerebellar cases a history of "fits" was occasionally obtained from patients or their friends, but it generally appeared on examination that these attacks were merely those of vertigo of the severer form, with clouding of consciousness. In one of their cases the authors observed a long series of peculiar seizures. The face was unaffected and respiration was undisturbed. Both arms were rigid and resistive to passive motion. The spasm was purely tonic in all limbs, and uniformly maintained for about three minutes. At the onset of the fit the eyes were in conjugate deviation to the right, but in its later stages they rolled slowly from side to side. The fit is ascribed to an irritative lesion of the cerebellum, and in this particular case reported was due to recent hemorrhage.

Charles L. Dana[1] also described a cerebellar seizure, sometimes occurring in tumor of the cerebello-pontine angle. The fits occurred in the day-time when the patient was awake, but once or twice they came on while she was sleeping at night. As the disease progressed they became more severe until, toward the end, they were always accompanied by loss of consciousness and a stiffening and irregular tonic spasm of the limbs, but without any actual convulsions or forced movements. She would suddenly fall down and lie unconscious in a sprawling attitude, such as one would expect in a person with an injured cerebellum. Dana regards this cerebellar syndrome as not infrequent. In tumors of the cerebello-pontine angle the syndrome is characterized by: (1) "Loud, high-pitched tinnitus or roaring and crackling noises, suddenly increased in intensity.

(1) N. Y. Med. Jour. and Phila. Med. Jour., Feb. 11, 1905.

(2) Vertigo, usually objective, and with or without forced movements. (3) A tendency to drop or fall in one direction or another instantly to the ground. (4) Sometimes sudden blindness and loss of consciousness. (5) In severe attacks, tonic spasms generally of an extensor type, lasting from one or two to five or ten minutes." Dana remarks that he has seen the cerebellar fits also in a case of hereditary cerebellar defect.

Cerebellar Gait. Andre Thomas[1] calls attention to the fact that disturbances of equilibrium do not belong to disease of the cerebellum alone; they also occur in lesions of the medulla when they involve cerebellar pathways. A case is described, to which the accompanying figure refers. In 1886 the patient's illness began as a progressive hemiplegia, with a crossed third nerve paralysis. The hemiplegia grew better, but the paralysis of the motor oculi persisted. In 1897 there appeared vertigo and disturbances of equilibrium, with paralysis of the fifth nerve on the left side, and intentional tremor of the arms, especially of the right. In 1900 the disturbance of equilibrium became aggravated and the legs became paretic. There was also a left facial paralysis. The cranial nerve involvements suggested a ponto-medullary lesion; the gait was typically cerebellar, as the figures show. Multiple sclerosis was regarded as a possibility, but the persistence of the paralysis of the fifth and third rendered this unlikely. Post mortem examination showed several lesions: a focus of medullary softening, a few foci of insular sclerosis, and disseminated meningitis. The medullary softening and the secondary degenerations in the cerebellum explained completely the disturbances of equilibrium and the tendency to fall to the left. The case is interesting because it shows how certain medullary lesions may resemble cerebellar ones.

Andre Thomas[2] also reports a typical cerebellar gait as occurring in lamellar atrophy of Purkinje's cells. The gait is well shown in the accompanying figure 8, and suggested a cerebellar lesion. On the other hand, the intention-tremor of the lower limbs, the hypotonus, the Romberg's sign suggested another site. Multiple sclerosis was, therefore, looked upon as the most probable cause of the

(1) Revue Neurologique, 1905, Jan. 15.
(2) Ibid., Sept. 30, 1905.

Fig. 7.—Instantaneous photographs showing the disturbances of equilibrium in walking, the broadening of the base of support, the uncertainty and the adduction of the upper limbs, especially the left. The last photograph of the series shows the patient during an attack of vertigo.—Revue Neurologique, Jan. 15, 1905, p. 10.

Fig. 8.—Instantaneous photographs showing disturbances of equilibrium exactly like those ordinarily found in gross cerebellar disease. Cf. with Figure 7.—Revue Neurologique, Sept. 30, 1905, p. 919.

symptoms, yet on post mortem examination no trace of it could be found. Ordinary examination of serial stained sections disclosed nothing. It was only after having studied some sections stained by picrocarmin that it was, almost accidentally, discovered that a large number of the Purkinje cells had disappeared. The same alteration was then looked for and found in the cerebellum of a tabetic, and in the cerebellum of one who had died with multiple sclerosis. The absence of all degeneration in the medullary substance is hard to account for. In this case Babinski's symptoms of diadocokinesia and asynergy were absent.

Encephalitis Cerebelli. F. Taylor[1] reports a case, the symptoms being tremor and ataxia of cerebellar type, nystagmus, absence of optic neuritis, following whooping-cough, and resulting in recovery after a duration of more than three and a half years.

MID-BRAIN.

William G. Spiller[2] in his Presidential address at the annual meeting of the American Neurological Association discusses the importance in clinical diagnosis of paralysis of associated movements of the eye-balls, especially of upward and downward movements. His extensive monograph is based upon nine of his own cases and thirty-eight others gathered from the literature. He devotes very little attention to the study of paralysis of lateral associated movements, these having been considered by him in 1903. The evidence is strong that that form of paralysis is indicative of a lesion of the posterior longitudinal bundle near the sixth nucleus, and is therefore a pontine lesion. The external rectus muscle on the side of the lesion may be more affected than the internal rectus of the other eye.

The disturbances of upward or downward associated movements have been studied less than those of lateral associated movements. Spiller is not of the opinion that the cortical center for such associated movements is yet known, though he admits that from our knowledge of a

(1) Lancet, Nov. 19, 1904.
(2) Jour. of Nervous and Mental Diseases, July and August, 1905.

probable cortical center in the frontal lobe for lateral movements, one probably exists for upward and downward movements. Hysteria as a cause of such paralysis of associated movements also doubtless exists. As a matter of fact, however, all the pathologic evidence that Spiller was able to obtain in cases of persistent palsy of associated upward or downward movement is indicative of a *lesion near the aqueduct of Sylvius,* in the mid-brain. It is extremely doubtful whether a lesion confined to the corpora quadrigemina and causing no pressure on the surrounding parts ever causes paralysis of associated ocular movements. A lesion developing ventrally on the *pons* may by pressure on the posterior part of the oculomotor nuclei cause paralysis of associated upward movement.

In Spiller's study of his nine cases and thirty-eight from the literature he finds that: (1) paralysis of upward associated movement without paralysis or paresis of downward associated movement was found in twenty-six cases. (2) Paralysis of upward associated movement with paralysis or paresis of downward associated movement was found in sixteen cases. (3) Paralysis of upward associated movement with impairment of lateral movement, often developing later, was found in fifteen cases. (4) Paralysis of upward associated movement without impairment of lateral movement was found in twenty-two cases. (5) Paralysis of downward associated movement without paralysis of upward associated movement was found in five cases. (6) The reaction of the iris was found to be impaired in fourteen cases, but in many reports no statements concerning it are made. (7) The optic nerve was found affected in fifteen cases. (8) Convergence was impaired in fifteen cases. (9) Ptosis was found in seven cases. (10) In all the cases except one, parts about the aqueduct of Sylvius were implicated.

As a result of his studies Spiller believes "persisting paralysis of associated *lateral* movement indicates a lesion of the posterior longitudinal bundle; that persistent paralysis of associated upward or downward movement indicates a lesion in the vicinity of the oculomotor nucleus and that paralysis of the associated ocular movements is not the result of a lesion of extracerebral nerve fibers. Lesions of the cerebral cortex may certainly cause paralysis of lateral

associated ocular movements, and possibly of upward or downward associated ocular movements, but cortical paralysis of associated ocular movements is transitory, unless possibly where the center on each side of the brain is destroyed. Paralysis of associated ocular movements may be caused by hysteria. Any case in which associated ocular palsy is persistent and is of organic nature is *unsuitable for operation* unless the operation is merely palliative, as the lesion is probably within the posterior part of the pons or cerebral peduncle, according to the form of the associated palsy, or else causes much pressure upon the dorsal portions of these structures. The paralysis of associated ocular muscles may be produced by inflammatory lesions or lesions of a similar character (alcohol, syphilis) as well as by tumor, and may disappear later in the course of the disease. Syphilitic ependymitis and cellular infiltration must always be considered in diagnosticating the lesion causing paralysis of associated ocular movements."

Ettore Gruner and Mario Bertolotti[1] reports two cases of associated upward and downward movements of the eyes, with perfect preservation of lateral movements. This article appeared too late for the two cases recorded to be included in Spiller's paper, and he merely alludes to them in a footnote. In one of these cases the clinical observation alone was made; in the other a post mortem examination was also obtained. The accompanying figures. A, B, C and D of Plate VI, excellently illustrate the ocular symptoms. The wrinkled forehead makes evident the ptosis and its fairly complete compensation. In Fig. A it will be observed that the eyelids, despite the patient's endeavors to raise them, are merely brought up to a very slight extent. Fig. B shows the absolute inability to raise the eyeballs, and the same inability to lower them exists. Figs. C and D show how easily lateral movements are made. In this case a small tuberculoma was found, practically replacing the central gray matter of the aqueduct of Sylvius, and involving a portion of the tegmentum, and all of both of the oculomotor nuclei. On the right side the posterior longitudinal bundle was crowded forward and many of its fibers were missing. The cells of

(1) Nouvelle Icon. de la Salpêtrière, March, April, 1905.

A

B

Plate VI.—Paralysis of upward and downward associated ocular movements.—Nouvelle Iconographie de la Salpêtrière, March-April, 1905.

the locus niger were discolored slightly on the left. The location of the tumor very accurately coincides with the position assigned to it by Spiller in his paper.

DISEASES OF THE SPINAL CORD.

LOCOMOTOR ATAXIA (TABES DORSALIS).

Pathology. Carl Weigert[1] calls attention to a change in the molecular layer of the cerebellum present in all cases of tabes dorsalis in which staining of the neuroglia is possible. In certain portions of this layer the neuroglia is heaped together more closely than normal, so that it is noticeable even to the untrained observer. Often, too, the individual neuroglia-fibers are thicker than normal. In combined general paralysis and tabes the changes are more pronounced than in tabes alone. They indicate a destruction of nerve-tissue and a substitution for it of neuroglia. The same changes are found in ischemic or hemorrhagic processes, in alcoholics, in syphilitics, and in the vicinity of solitary tubercles.

Edinger[2] once more discusses the "exhaustion diseases" of the nervous system in an extensive series of papers. He remains committed to his original hypothesis that tabes dorsalis is a disease of exhaustion. When the demands made upon a nerve-cell are greater than can be met by its assimilative capacity, the cell undergoes simple atrophy. There are three types of diseases of the nervous system: the toxic diseases, the focal diseases and the exhaustion diseases. In the latter, lost nerve-tissue is replaced by neuroglia, and the whole process is a progressive one. Toxic conditions may hasten the advent of exhaustion diseases; thus the metal lead first affects muscles most used by the painter, viz.: the extensors of the wrist. Syphilis and alcohol also act as does lead. Some exhaustion diseases affect nerve-tracts which were never properly developed; hence, they atrophy prematurely, and to this category belong the hereditary nervous diseases, such as juvenile tabes,

(1) Neurolog. Centralblatt, Aug. 16, 1904.
(2) Deutsche med. Wochenschrift, 1904, pp. 1633, 1800, 1921; 1905, pp. 4 and 135.

many of the combined scleroses, etc. The therapeutic suggestion is obvious; the tabetic patients should walk very little, take but little exercise, go to bed every few days, and rest a great deal.

Symptomatology. Carl D. Camp[1] calls attention to *muscular atrophy,* present in from 15 to 20 per cent of cases. In the case reported there was muscular atrophy in the lower extremities, and trophic disturbances in the distribution of the fifth nerve, as indicated by the painless falling out of the teeth. Anatomically, changes were found in the posterior columns and posterior roots, together with marked degenerative changes in the nerve-cells of the anterior horns, and partial degeneration of the fifth nerve. A second case like the first is added, and the literature of muscular atrophy in tabes is carefully reviewed. Medea[2] reports the case of a tabetic showing unusual atrophy with contractures in the left upper extremity. There was no post mortem examination. A much more interesting case of the same sort is one reported by Marie and Léri.[3] This patient showed atrophy of the muscles innervated by the motor root of the left trigeminal nerve, with concomitant blindness and paralysis of the third, fourth, the sensory portion of the fifth and sixth pairs of cranial nerves. The eyeballs were immovable, and there was double ptosis. No post mortem is recorded. Marie's diagnosis was a basal meningitis.

Febrile Forms of Tabes. M. Faure[4] describes two febrile forms in tabes, one due to febrile accidents, such as infection of cavities, particularly the pelvis, or of the intestine, or of a few large bronchi; (2) and a febrile form of tabes. The latter occurs in one of two types: (1) In the first type there are febrile attacks, the temperature ascending to 100.4 or 102.2 degrees F. daily. The course of such a case is like that of a subacute or chronic infection, and the temperature curve is irregular. Such forms are of grave import, often leading to death within two years. Sometimes, on the other hand, the fever subsides and the patient becomes as an ordinary tabetic. (2) The second

(1) Univ. of Penn. Medical Bulletin, January, 1905.
(2) Revue Neurologique, July 30, 1904.
(3) Revue Neurologique, Feb. 28, 1905.
(4) Progrès Médical, 1905, p. 103.

form shows a continuous fever without intermission, the temperature rising only a few tenths above the normal. At the least exertion the fever rises higher, and the patient is in an asthenic condition.

Tabetic Blindness. Andrè Léri[1] in a long article on this subject concludes that the particular variety of blindness, even though it has associated with it but a small number of spinal symptoms, is really a localization at the level of the base of the brain of the same morbid process as obtains in general paralysis and true tabes dorsalis. Clinically, this form of "tabes-blindness" is not alone accompanied by slight spinal symptoms, but also by slight encephalic ones; and pathologically the lesions underlying these are those usually met with in general paralysis. Léri believes that "tabes-blindness" is as near a relative of general paralysis as is tabes dorsalis itself. Tertiary syphilitic amaurosis, called "tabetic," may therefore be associated with the spinal symptoms of tabes or the cerebral symptoms of general paralysis.

The blindness comes on in two stages. The first, which is relatively acute and inflammatory, lasting from a few months to two or three years at the most, is distinguished anatomically by an intense vascular new-formation; clinically, by a rapid disappearance of vision first in one eye, then in the other, and by violent headaches as a rule frontal and orbital. Often there are color sensations denoting irritation of the optic nerve. The second stage, properly a stage of sclerosis and slow atrophy, lasting from three or four years to ten, twenty or thirty years and more, is marked anatomically by the obliteration of the pre-existing and newly-formed vessels, and clinically by the extremely prolonged persistence of the notion of day and night, and of light. In this stage the symptoms of irritation disappear, but the mental symptoms sometimes persist when the lesions of general paralysis are present.

In another article on the relations between blindness and general paralysis and tabes, Léri[2] affirms that blindness is usually regarded as common in tabes, and rare in general

(1) Gazette des Hôpitaux, July 30, 1904.
(2) Progrès Médical, 1905, p. 124. See also Revue Neurologique, Aug. 30, 1904, and Nouvelle Iconographie de la Salpêtrière, 1904, pp. 304 and 358.

paralysis; and that if optic atrophy exists in tabes the remaining tabetic symptoms are fairly benign. On the contrary, the real facts are as follows: (1) As regards general paralysis, blindness is rare when the mental troubles are grave, but slight disturbances of vision are by no means rare. Moreover, blindness has often been observed *before* the onset of the mental symptoms. (2) As regards tabes, blindness is rare in severe cases and is frequent only in those cases where the symptoms are mild, resulting from lesions of the posterior cord; (b) when blindness does appear it is usually the first sign or among the first symptoms of tabes; (c) the form known as "tabes-blindness" [above mentioned—ED.] is accompanied not alone by cord symptoms but also by slight mental defects analogous to those of general paralysis. In general this latter form might better be called "paralytic" optic atrophy. All three of these diseases—tabes dorsalis, general paralysis and "tabes-blindness"—represent simply *different localizations of the same process,* probably resulting from tertiary syphilis.

Green and violet vision in tabes is described briefly by L.Bregman,[1] being regarded as symptoms of irritation of the second nerve. A case is reported of a man 36 years of age with a luetic history.

Other Symptoms. Adam Loeb[2] described a case of interruption of respiration in tabes in a man 35 years of age brought to the clinic suffering from a most severe gastric crisis, with what looked very like coffee-ground vomitus, mingled with bile. At first *ulcus ventriculi* was suspected and 1 centigram of morphin was given hypodermically. Shortly thereafter the patient's arm in which the injection was made began to jerk, breathing became reduced in rate, and finally stopped altogether, very deep cyanosis supervening. Traction of the tongue brought back the breathing, and consciousness quickly returned. It was then discovered that he had had a specific chancre five years before and that he was suffering from a gastric crisis and not from collapse from perforation of an ulcus ventriculi. This was not a case of laryngeal crisis, but seemed to be due to some effect exerted upon the respiratory center. A similar

(1) Deutsche Zeitsch. f. Nervenheilkunde, XXVI, Heft 4-6.
(2) Muench. med. Wochenschrift, Oct. 11, 1904.

case is reported by McBride. Oppenheim and Siemerling also describe disturbances of respiration in tabes.

Idelsohn,[1] working in Pierre Marie's laboratory, describes very fully the pathology and histology of the tabetic foot, giving an excellent illustration of the bony deformities of a patient dying at the age of 73, having had tabes approximately twenty-five years. The cord showed the characteristic tabetic findings. Frequently the tabetic foot is associated with flat-foot.

Balint[2] reports dermographia in a tabetic 60 years of age, the irritated skin first showing pallor, then diffuse redness, and then rising up 5 to 6 mm. above the surrounding skin. The disturbance lasted about ten to twelve minutes, then began to fade and was gone in two hours. So marked a vasomotor disturbance in tabes, so generally distributed over the body, is regarded as rare by Balint.

Treatment. H. S. Frenkel[3] discusses the fundamental principles underlying his exercise-treatment for tabes. It is directed towards one symptom, namely, the disturbance of co-ordination. In walking the back muscles are made much use of, and hence the importance of exercises for moving the trunk as in walking. Often the back is stiff or the hip-joints are stiff, or the patient cannot hold the trunk in equilibrium. If in addition to the disturbance of co-ordination of the back muscles the lower extremities be ataxic, the patient is unable even to stand. Various degrees of kinetic and static ataxia are mentioned and discussed. Unfavorable complications preventing exercises are attacks of pain lasting several days, various crises, marked acceleration of the heart-action and pronounced hypotonia with stretching of the joint ligaments. Massage of the muscles is not countenanced.

On the other hand Determan,[4] who regards tabes in the light of Edinger's teachings as an exhaustion-disease, declares that over-exertion will bring out a latent tabes. In early cases he would use mercurial inunctions, but if they do not avail he would at once put at functional rest that part of the body in which the first tabetic symptoms appeared. Thus a better state of nutrition of the central cells

(1) Deutsche Zeitschrift für Nervenheilkunde, XXVII, p. 121.
(2) Neurolog. Centralblatt, 1904, p. 917.
(3) Berliner klin. Wochenschrift, June 5, 1905.
(4) Deutscher med. Woch., April 6, 1905.

may be encouraged and forced feeding should be used as an adjuvant. Lying quietly in the open air is excellent treatment. Short walks of measured amount may be permitted.

Friedr. Schultze,[1] after discussing the early symptoms and diagnosis of tabes, advises at first a rigorous antisyphilitic treatment with mercury and potassic iodid. The mercurial course may be repeated in six to twelve months. Electricity, baths and massage are worthless, and there are no internal remedies. Baths in natural carbonated waters at the springs, and not in one's home, prove an exception to the rule; they may be of service, but the tabetic is to understand that his lancinating pains do not constitute rheumatism, and that he is not to take hot baths. On the other hand, he is to take no cold plunges. The patient must be handled very gently. Suspension, electricity and even massage are justified if the patient is in danger of taking up with morphin in his despair of being relieved of pain. Weakness of the bladder sphincter and sexual weakness may be helped with strychnin or nux vomica. The gastric crises are especially hard to influence, and the use of morphin is hard to avoid. Codein, dionin and different somnifacients should first be tried. For the ataxia Schultze favors the Frenkel exercises, but many of these can be taken in the recumbent posture.

Oberthur and Bousquet[2] call attention to sodic nitrite, used in Italy by Petrone, and in Austria by Winternitz and Pal. For the pains it is very efficacious and should be the remedy of choice. Moreover, it aids co-ordination and re-education becomes much easier. It acts best when given hypodermically.

Guy Hinsdale[3] reports an encouraging case of locomotor ataxia, the patient showing remarkable improvement in a short space of time. He was put to bed and given the usual rest treatment, with massage, sinusoidal electricity and educational movements. In eight weeks his weight increased from 108 to 144 pounds, his pains left him, his gait improved and he returned to his usual occupation. The rest treatment is recommended.

(1) Deutsch med. Woch., Nov. 24, 1904.
(2) Progrès Médical, 1905, p. 135.
(3) Jour. Am. Med. Assn., Feb. 18, 1905.

MULTIPLE SCLEROSIS.

Diagnosis. P. Morawitz[1] calls attention to the recent monograph of E. Müller upon multiple sclerosis, based upon eighty cases observed in Strümpell's clinic at Erlangen and Breslau. The form described by Charcot, of which the cardinal symptoms are nystagmus, intention tremor, and the scanning speech, is by no means the usual one in the rural districts of Germany. Indeed, in its early stages these symptoms are very much in the background, and a large number of other symptoms, very like those of hysteria, take their place. The improvements in the spastic-paretic phenomena or the ataxic disturbances are very confusing. There is no classic form of multiple sclerosis, the classic symptoms being all present in only 15 per cent of Müller's eighty cases. The cases are rather to be divided up into a cerebral, a bulbar and a spinal type. Morawitz has investigated the material at the Tübingen clinic, consisting of thirty-three cases, and has obtained results substantiating Müller's. Only three of the cases came to post-mortem examination, and so permitted a positive confirmation of the diagnosis. During the same period at the same clinic there appeared twenty patients with tabes dorsalis and ten with cerebrospinal syphilis. In rural districts, therefore, multiple sclerosis is the most frequent of the organic diseases of the central nervous system, exactly as claimed by E. Müller.

Of the thirty-three patients, eighteen were females and fifteen males. The disease began before the age of 20 years in seven individuals, between 20 and 30 years in fourteen individuals, between 30 and 40 years in ten individuals, and after 40 years in two individuals. The earliest onset was at the age of 8 years.

As to etiology, Strümpell suggests an endogenous cause rather than the infections, intoxications and metallic poisonings usually alleged. In 75 per cent of Strümpell's cases and 70 per cent of Morawitz's cases no exogenous cause could be found. In only 9 per cent of the cases was there a neuropathic heredity.

As to symptomatology, most of the cases showed atypical forms, the so-called *formes frustes* of Charcot. Least

(1) Deutsche Archiv f. Klin. Med., Dec. 12, 1904.

important of the cardinal symptoms is the scanning speech, present only four times in thirty-three patients. In three very advanced cases confirmed by post mortem examination this symptom was absent. In eight cases defects of speech were present, consisting of slow, monotonous speech without modulation. There were also observed patients with dysarthria, syllable-stumbling, or nasal speech as in bulbar paralysis. Nystagmus was present in seventeen, or 50 per cent of the patients, and in many of these it was only slightly marked. Intention tremor, according to Müller, is absent in most of the early cases and is present in 75 per cent of the late ones. Of the thirty-three cases of Morawitz, twenty-three, or 70 per cent, showed ataxic movements and only fourteen, or 42 per cent, showed true intention tremor. Curiously enough, the symptom was present in only one of the three cases examined post mortem.

More important than the classic symptoms are ataxic disturbances; eye symptoms, especially optic atrophy; and spastic paresis of the lower extremities with preservation of the sensibility and cutaneous reflexes. In most of the cases the anamnesis is the same; they complain of gradually increasing weakness and clumsiness in the legs, and then in the arms. Then vertiginous attacks follow, with various paresthesias. The latter may antedate all other symptoms by more than a year. In a third of the patients bladder disturbances are complained of early. Either the urine cannot be properly held or there must be a good deal of urging in voiding it. Disturbances of defecation are less common, the mishaps occurring only when the fecal matter is very thin. Very characteristic are the spontaneous improvements and even disappearances of troublesome symptoms. Later, complaints as to tremor and gait are made. At least 70 per cent of the patients complained of sensory disturbances of a subjective sort, and in at least 60 per cent complaints were made of vesical disturbances. Atrophy of the optic nerve was found in 40 per cent of the patients, yet visual disturbances were complained of in but two cases. Inequality of the pupils was observed eight times. As a rule, the tendon reflexes are uniformly increased. The abdominal and cremasteric reflexes, on the other hand, are absent in more than half the cases, and in 75 per cent of the cases they are not normal, being weak

or present on one side and absent on the other. In eighteen cases where Babinski's toe-sign was tested it was positive eleven times.

Besides these signs there is an evident impairment of the intellect or a loss of memory. In only two cases was forced laughter found. In two of the three cases examined post-mortem, contractures in the lower extremities existed, associated with fibrillary twitching and cramps in the calves. Muscle atrophy was present in one case.

Hobhouse[1] declares multiple sclerosis to be the most common of the diseases of the central system. In five years he has seen nine cases in his out-patient department against six of paralysis agitans, five of tabes dorsalis, two or three of general paralysis, four of cerebral tumor, and a few odd cases of syphilis of the cord, progressive muscular atrophy, etc. Many cases escape diagnosis because they are dubbed "hysteria."

The symptoms are so diversified that patients suffering with the disease may appear before a surgeon, gynecologist, aurist, laryngologist or ophthalmic surgeon. Great as was Charcot's service it has kept down a proper understanding of the symptomatology because of the stress laid by him upon the so-called classic symptoms which are the ones least frequently present. If the diagnosis is to be made only when the classic triad is present it will be seldom made. Indeed, we must be content to find but one or two of these symptoms present. The majority of cases begin in one or two or three different ways. Most patients go first to the physician complaining of weakness in the legs of one or both sides, or of nervousness and trembling, but a considerable percentage go first to the oculist. Hobhouse then describes sixteen of his own cases, his statements closely coinciding with those of Morawitz.

Most of the patients in their early stages are regarded as hysterical, because of the transitory nature of the symptoms. Indeed, it is this which is really typical. The patients can see one day and be almost blind the next, or they can walk fairly well one moment and the next be unable to move from their beds. The symptoms may come on with startling suddenness and depart almost equally

(1) Lancet, Feb. 18, 1905.

suddenly. Ultimately the transitory difficulty in walking becomes permanent and contractures follow, or the tremor now and then present becomes the typical intention tremor. Many of the patients, too, have a typically hysterical manner, being emotional and easily moved to tears or laughter. In general, the slower the onset of the symptoms the more hopeful is the prognosis as to remissions. The fact that the earliest symptoms are cerebral does not necessarily imply that those will be specially predominant later.

Dercum and Gordon[1] report a case of multiple cerebrospinal sclerosis, calling attention to the fact that it is supposed to be rare in this country, doubtless because American physicians insist upon the classic picture before making the diagnosis. According to Dercum and Gordon only six cases with post mortem examination have thus far been reported in this country, two by Spiller and Camp, one by Burr and McCarthy, one by Hunt, and two by Spiller. Therefore, this is the seventh case and concerns a white woman 29 years of age. She complained of pain and tenderness in the pelvis and profuse leucorrhea. She has had three miscarriages and had borne no living children. Three years previous to admission she fell and struck on the buttocks. Operation showed cystic ovaries. After recovery she was sent to the nervous wards. The body was emaciated and showed distinct atrophy of individual groups of muscles, especially the thenar and hypothenar. There was complete loss of power in the lower extremities. She could not flex or extend her legs. There was double foot-drop; the left foot was rotated inward. The knee-jerk was increased on both sides; ankle-clonus existed on the right, and the toe-sign was present on both sides. She was hyperalgesic everywhere and complained of pain in her shoulders and hips. There was a very coarse intention tremor, more marked on the right than on the left. The speech was distinctly scanning. Lateral nystagmus was present in both eyes. The pupils were unequal, the right larger than the left; they responded to accommodation, but very little, if any, to light. The left lid showed slight ptosis. The retinæ were not examined. There was incontinence of urine and feces. A large bed-sore was pres-

(1) American Journal of Medical Sciences, 1905, p. 253.

ent over the sacrum. Later the knee-jerks began to disappear.

Post mortem there was found in the sacral portion of the cord to be an entire absence of the gray matter. It was preserved in the lumbar cord and was affected in the dorsal and cervical portions. The white matter was destroyed at various points, the maximum destruction being in the cervical region. In the medulla the sclerotic process was especially irregular, but the gray matter suffered more than the white. An interesting feature was secondary degeneration, which is a rare occurrence in multiple sclerosis. Attention is called to the disappearing knee-jerks, at first exaggerated and finally absent, Marie and others believing that the knee-jerks are never absent. Interesting, too, was the involvement of the third, fourth and sixth nerves, with their nuclei, and also of the optic tracts.

Rudolf Maier[1] reports a peculiar case of circumscribed multiple sclerosis, the pathologic process being limited to the cervical cord. The exaggeration of the knee-jerks suggested a lateral tract sclerosis, and the spastic contractures, absence of sensory symptoms and atrophies seemed to confirm that diagnosis. On the other hand, the violent headache, neck-rigidity, somnolence, slowness of speech and thought, the incontinence of feces and urine, all suggested hydrocephalus internus. Post mortem, both multiple sclerosis and hydrocephalus internus were found.

P. Lejonne[2] in an extensive paper carefully describes the so-called "amyotrophic form of multiple sclerosis," that form in which muscle atrophy is a feature. As a rule, cases showing the atrophy are severe ones and the patients therefore show severe trophic and sphincteric disturbances and marked mental impairment. The muscular atrophy does not differ from that seen in systematized poliomyelitis or amyotrophic lateral sclerosis. The course of these cases is rapid, as a rule affecting all four limbs and being followed fairly early by sphincteric and trophic disturbances, particularly bed-sores. [The case of Dercum and Gordon evidently belongs to this type.—ED.]

(1) Prager med. Woch., Nov. 10, 1904.
(2) Gazette des Hôpitaux, 1904, pp. 1,097 and 1,105.

Pauly[1] reports a case of intention tremor stopped by alcohol. When the patient was sober the tremor was severe; when under the influence of alcohol the tremor subsided.

DISEASES OF PERIPHERAL NERVES.

MULTIPLE NEURITIS.

S. Auerbach[2] discusses the *pathogenesis* of multiple neuritis. Ordinarily multiple neuritis caused by alcohol affects the distal portions of the nerves of the extremities, the proximal portions escaping. Auerbach reports the case of a waiter suffering from multiple neuritis, in whom the left sterno-cleido-mastoid, both trapezii and deltoids, were more affected than other portions. He shows that the posture of this waiter in attending evening functions, where movements of the body and head and neck were necessary in bending over chairs, had much to do with the location of the lesions. He is an ardent advocate of Edinger's exhaustion theory and ingeniously applied it to the case reported.

H. Waldo[3] reports a case of peripheral nerve intoxication in a young man of good physique 33 years of age, with a tendency toward alcoholic beverages. His illness began with retching and severe pains in the legs lasting all one night. In three days' time there was total loss of power in both legs and partial loss in the arms. Then the pains left him, only tenderness in the legs and finger-tips remaining. A catheter had to be passed twice daily for ten or twelve days. Waldo prefers the appellation "nerve-intoxication" to neuritis, which denotes inflammation, and he is unwilling to admit that degeneration of the nerves exists in the patient. The electrical reactions obtained were said not to be characteristic of degeneration since response both to faradism and galvanism was lost.

Wharton Sinkler[4] reports some uncommon forms of multiple neuritis, including four of puerperal origin. By far

(1) Lyon Méd., Nov. 27, 1904.
(2) Muench. med. Woch., Aug. 16, 1904.
(3) Lancet, Dec. 10, 1904.
(4) Jour. Am. Med. Assn., Feb. 25, 1905.

the most common variety is that due to alcohol, first described by Samuel Jackson, Sr., of Boston, as alcoholic paraplegia in 1822. It is likely that patients leading a sedentary life are more liable to alcoholic neuritis than persons actively employed. Hence, the greater frequence of alcoholic neuritis in women. Another form becoming quite frequent in large cities is the result of coal-gas poisoning. Carbon disulphid is also a severe causal factor and is found chiefly in workers in rubber. Metallic poisons, especially lead, copper, phosphorus and mercury, are potent factors. Arsenical neuritis due to Fowler's solution is rare, a case being here reported in a child 7 years of age. The English epidemic of 1899 due to arsenic in beer is alluded to. Multiple neuritis is often a sequel of diphtheria, grippe, typhoid and malarial fevers. That it may be a sequel of puerperal septicemia is shown by four cases here reported. Even rheumatism and gout may lead to it.

G. E. Price[1] reports a case of malarial infection presenting symptoms of peripheral neuritis in a girl 8 years of age. A blood examination showed the plasmodium to be present, and with the administration of quinin the symptoms rapidly cleared up. The chief complaints were dragging the feet in walking, a feeling of pins and needles in the extremities, and severe pain in her right leg and arm. About a month before she was first seen a tremor in both hands developed. There was marked weakness in the flexors of both ankles, most marked on the right side. There was no muscular atrophy, no pain or tenderness over the nerve trunks. The knee-jerks could be obtained only feebly by reinforcement. Sensation was unaffected. [The symptoms as recorded are not such as wholly to justify a diagnosis of multiple neuritis.—ED.]

Edens[2] calls attention to the very few cases reported of multiple neuritis as a sequel of measles, the only ones on record so far as he knows being one reported by Morton and one by Monro, both of which are summarized. Edens, therefore, feels justified in reporting a case at length following a very severe attack of measles in a girl 16 years of age.

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(1) American Medicine, June 3, 1905.
(2) Berliner klin. Woch., Aug. 8, 1904.

Jelliffe[1] has carefully searched the literature, though not exhaustively, without finding a single recorded case of multiple neuritis following the use of wood alcohol as a beverage. Of this affection, however, Jelliffe has seen three cases, their histories being very briefly recounted, the first of the three being that of a man who was sold a "tax-free (moonshine) whisky" for 25 cents per gallon, it really being a 35 per cent wood alcohol with suitable flavors to make a "richly blended article." Two other cases occurred in varnishers who did nothing but shellac work, the shellac for the most part being dissolved in wood alcohol. They were exposed to the evaporating fumes for long hours and they worked in a small room. Both suffered from the hyperesthesic form of multiple neuritis. There was distinct motor weakness, however, in both instances.

P. N. Gerrard[2] reports seven cases of beri-beri with post-mortem examination, all being fulminating cases. The first case was that of a Chinaman 53 years of age, whose first bad symptom appeared only four days before his death. Post mortem there was found a dilated stomach which was "congested with deep purple erosions for a considerable area in the neighborhood of the pylorus. It contained a mucoid fluid in which a bacillus 2.8 micromillimeters long by 1.05 broad predominated." The second case developed fulminating symptoms November 21, and a solution of trinitrin was administered with the object of relieving the right heart. "The stomach symptoms were severe, pain and fullness being both complained of, the latter being evidenced by the usual epigastric inflation and puffiness." The patient died at 1 a. m. on November 22, in less than twenty-four hours' time. Post mortem there was found "deep congestion of all the mucous membranes, an ounce of fluid was present in each pleura, there was deep general congestion of the lungs, the pulmonary artery was full of dark clot, the pericardium contained four ounces of fluid and the anterior surface of the heart showed punctiform hemorrhages. All the other organs were congested and the stomach exhibited a dull, reddish-purple interior with considerable erosion, which was most marked at the pylorus." The records of the five other cases are given, but no con-

(1) Medical News, March 4, 1905, p. 387.
(2) Lancet, June 17, 1905.

clusions are drawn as to the cause of the appearance of the fulminating type, occurring in a case apparently running the usual chronic course.

J. E. Donley[1] reports a case of toxic degeneration of the lower motor neurones, resembling in its symptoms both poliomyelitis, multiple neuritis and Landry's paralysis. The case occurred in a maiden lady 30 years of age, and the weakness complained of involved the hands, forearms, legs and feet. The case is correlated by the author with the group reported by Stanley Barnes (formerly reviewed in the Practical Medicine Series). The resemblance of these cases to peripheral neuritis is striking. There is a comparatively rapid onset, a symmetric distribution, tingling and numbness, tenderness over the muscles and nerves upon pressure, and distinct muscular atrophy. The author's conclusions follow: "(1) Toxic degeneration of the lower neurones, acute anterior poliomyelitis, peripheral neuritis and Landry's paralysis are essentially degenerative conditions of nervous elements. (2) The exciting cause of this degeneration is a toxemia, which may be the result of bacteria, autotoxins or poisons introduced from without. (3) The whole neurone, both cell and fiber, suffers in every case, the clinical symptoms, however, depending upon the intensity, the duration and the anatomical situation of the morbid process."

F. Terrien[2] reports a case of optic neuritis and atrophy in the course of an ordinary facial erysipelas, in itself alone benign. The optic neuritis resulted from a bilateral orbital cellulitis, with compression of the second nerve and complete loss of vision.

E. A. Shumway[3] reports a case of association of optic neuritis and facial paralysis, calling attention to the relative infrequency of such association. The case occurred in a girl of 19 years, appearing at de Schweinitz's clinic. In 1894 Hoffman described two cases of such association and de Schweinitz had reported two cases. In all seven cases were found in the literature. Shumway concludes as follows: "(1) Optic neuritis is occasionally associated with facial paralysis, either alone or as a part of a multiple

(1) Boston Med and Surg. Journal, June 29, 1905.
(2) Progrès Méd., Sept. 10, 1904.
(3) Journal Am. Med. Assn., Feb. 11, 1905.

neuritis; the etiologic factor may be rheumatism, but at times appears to be an infection the nature of which is as yet undetermined. The optic neuritis is usually of the retrobulbar type, but a decided papillitis may be present and be followed by more or less marked atrophy. In cases of multiple neuritis of the cranial nerves the eyegrounds should be examined for possible optic nerve complication. (2) In facial paralysis, flattening of the face and enophthalmus may appear, and are to be considered as due to a neuritis of the fifth nerve and not to involvement of possible sensory fibers in the facial nerve."

MISCELLANEOUS.

PROFESSIONAL CRAMPS.

P. Kouindjy[1] devotes a very interesting paper to the treatment of professional cramps by systematic massage and re-education. All sorts of cramps exist, such as writer's cramp, violinist's cramp, pianist's cramp, tailor's cramp, dancer's cramp, and Basedow in 1851 described a cramp caused from milking cows. The professional cramp is a kind of professional ataxia, and resembles the ataxia of tabes in that it does not become evident until movement is attempted. Hence, Kouindjy favors the appellation, *professional ataxia*. It may appear as a paralytic form, as a spastic form, as a tremulous form. It furthermore has within it a psychic element. Like tabetic ataxia, it begins with a slight inco-ordination, and utimately results in complete loss of power. Figs. 1 to 5 illustrate five specimens of writing. Figs. 1 and 3 are the handwritings of two accountants and illustrate the tremulous and the spastic type. Fig. 2 is the handwriting of a bank clerk and shows the paralytic type. Fig. 4 is the handwriting of a day laborer whose occupation is digging, and Fig. 5 is the handwriting of a day laborer doing miscellaneous work. In comparing these specimens one is struck by the perfect analogy between them. Fig. 4 is an example of the tremulous type and Fig. 5 of the spastic type. But

(1) Nouvelle Icon. de la Salpêtrière, March-April, 1905.

A

B

C

D

Plate VII.—Illustrating the treatment of professional cramps.—Nouv Iconographie de la Salpêtrière, March-April, 1905.

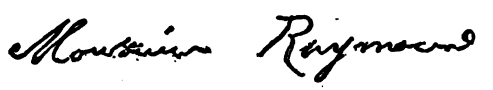


Fig. 1. — Type trémulant.

Fig. 2. — Type paralytique.

Fig. 3. — Type spasmodique.

Fig. 4. — Ecriture d'un terrassier.

Fig. 5. — Ecriture d'un journalier.

neither of these writers suffered from writer's cramp; they merely show the consequences of a defect in the education of the muscles used in writing.

All sorts of drugs, electricity, surgical intervention, and what not have been tried, to the exclusion of muscle-training and re-education of the muscles. Some recommend rest; others a change of occupation. If one examines carefully the hand of one suffering from writer's cramp, as a rule the flexors will be found contracted and their extensor antagonists paretic; hypertonia of the flexors and hypotonia of the extensors. The conclusion is obvious: to massage the hypotonic muscles and leave the others alone. Kouindjy begins by deep *effleurage*, longitudinal and circular, of all

the extensors of the hand. He then follows with kneading, first with one finger and then with several. This is succeeded by *tapotement* and a gentle *petrissage*. No roughness is to be tolerated. The interossei are massaged by the ball of the index finger. Mechanical vibration is also used.

This massage is followed by a series of exercises for the extensors, chiefly exercises with different weights, with a cane, etc. A string slipped over the end of a finger carrying a receptacle of any sort on its other end, into which receptacle various weights may be placed, is the only apparatus required. For diversity a light cane may be used. The figures of Plate VII illustrate the character of the exercises very clearly. Small weights are used, beginning with 50 grams and increasing to 500 (one pound). Four or five movements are made with each finger separately, the loop, of course, being easily removed from one finger. An exercise with a rubber ball of average size affords some relief from monotony. The ball is placed on the dorsal surfaces of the fingers and by the extensors is thrown into the air and caught, or it may be allowed to fall back upon the fingers again between the first and second fingers or between the thumb and index fingers, or between the middle and ring fingers. A coin can be used in the same manner.

The exercises with the cane are of two sorts, the first being exactly like the exercise with the ball and the second being illustrated in Figs. A and B. The cane is placed over the dorsal surface of the middle and ring fingers, and beneath the index and little fingers. The cane is then turned around by depressing the little finger and permitting the cane to slip out from under the first finger. At this stage the cane is held perpendicularly between the ring and little finger and then turned horizontally by the action of the third finger and the first finger and then placed beneath the opposite end. The cane is now in the position seen in Fig. B, supported on the dorsal surfaces of the index and little fingers and being covered by the middle and ring fingers. The patient should then rest, and then rotate the cane again.

These exercises should next be followed by exercises designed to re-educate the muscles. It is best to begin by teaching the patient to write backward, the position of the hand being shown in Fig. D. After backward writing is

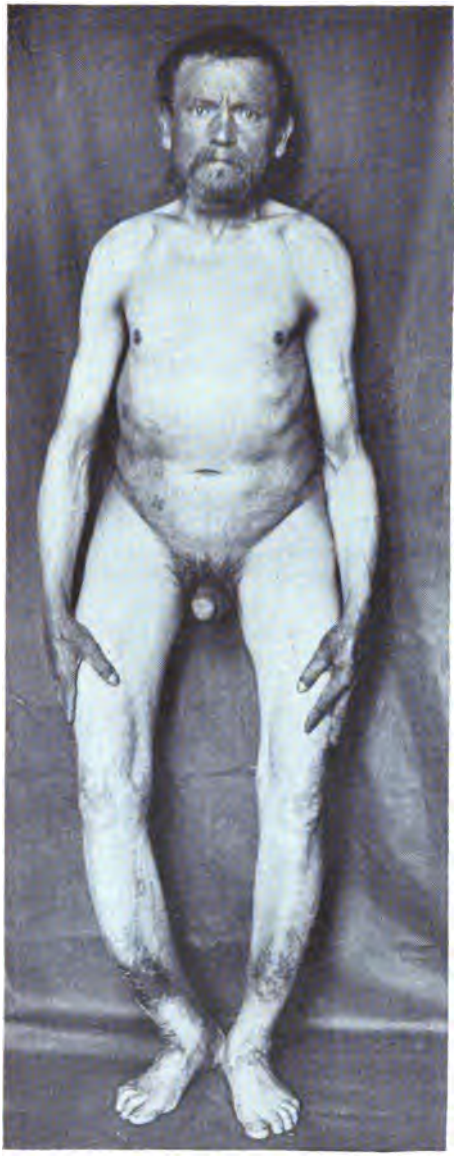


Plate VIII.—Paget's Disease.—Nouvelle Iconographie de la Salpêtrière, May-June, 1905.

Plate IX.—Paget's Disease in brothers.—Nouvelle Iconographie de la Salpêtrière, May-June, 1905.

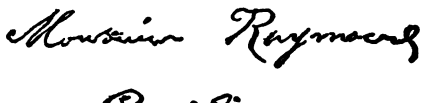


Fig. 9. — Avant le traitement.

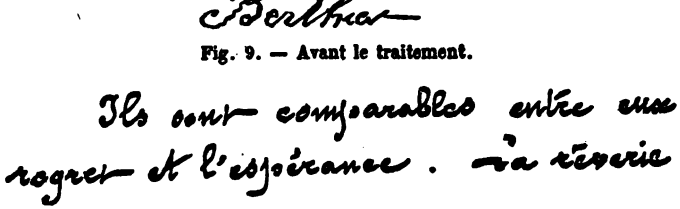


Fig. 10. — Après le traitement (écriture avec main renversée).

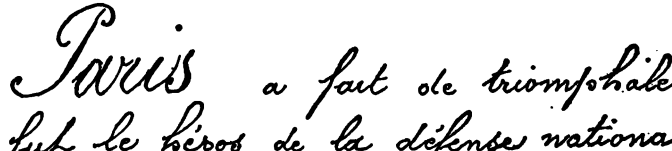


Fig. 11. — Après le traitement (écriture ordinaire).

CRAMPE DES ÉCRIVAINS (FORME SPASMODIQUE)

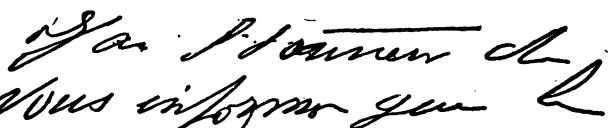


Fig. 12. — Avant le traitement.

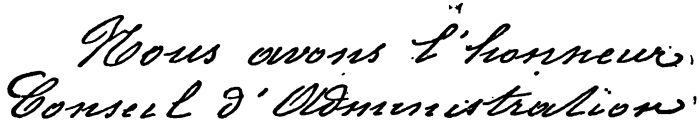


Fig. 13. — Après le traitement (écriture ordinaire).

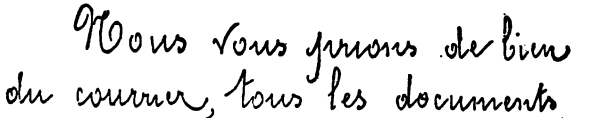


Fig. 14. — Après le traitement (écriture avec main renversée).

acquired, writing in the usual manner is next attempted In these exercises the principle of making haste slowly is paramount. Kouindjy has the patient dip his pen into the ink after every single stroke of the pen. In making the letter "a," for example, three strokes are used and ink is twice taken. The success of this method of treatment is illustrated in Figs. 9 to 14, inclusive.

Zabludowski,[1] who has frequently written authoritatively on this subject, suggests the use of a pencil-carrier, a piece of apparatus designed to hold the pencil, because in cases severe enough to make such an apparatus necessary the use of pen and ink is impossible. This apparatus, which he describes, can be made use of after hemiplegia, even if contractures have supervened. In those cases where the mere grasping of a pen or pencil brings on a cramp a wristlet of leather, also crossing the metacarpus, can be worn. The pen or pencil can be fastened to the index finger by a wide rubber ring without danger of constriction. In paralytic forms, where the hand and fingers refuse to functionate, or where there is swelling, an elastic rubber, surrounding the first and metacarpus may render effective service. In cases where pain is the cause of inability to write the treatment is educational. If the hand is properly held the pain will cease. For all these forms massage is also recommended.

PAGET'S DISEASE.

In 1876 appeared Paget's treatise on osteitis deformans, based on about 100 observations. Oettinger and Agasse-Lafont[2] report three cases, all belonging to the same family, and advance a new theory of pathogenesis. Several cases have already been published in which an hereditary element is found. Plates VIII and IX appended show the characteristic deformities. The cranium is large, the circumference being 61 cm. (24 2-5 inches) in one of the brothers. The bones of the face are normal. The clavicles are increased in size, the spinal column shows kyphosis, the arms are normal. The lower extremities are very much arched with the convexity antero-externally.

(1) Progrès Médical, 1905, p. 139.
(2) Nouvelle Icon. de la Salpêtrière, May-June, 1905.

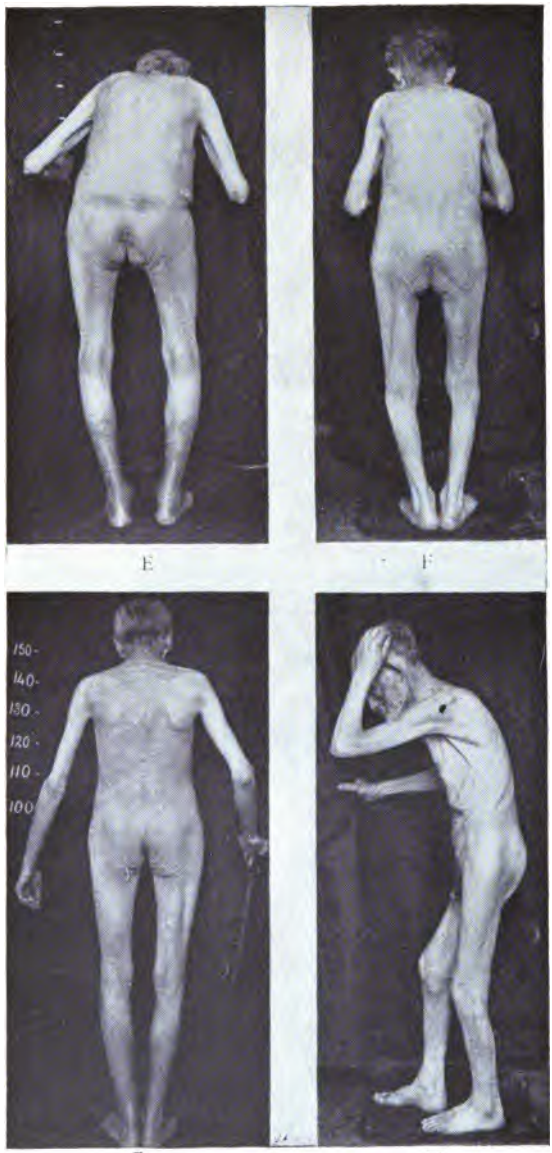


Plate X.—Senile deformities of the skeleton simulating Paget's Disease.—Nouvelle Iconographie de la Salpêtrière, Jan.-Feb., 1905.

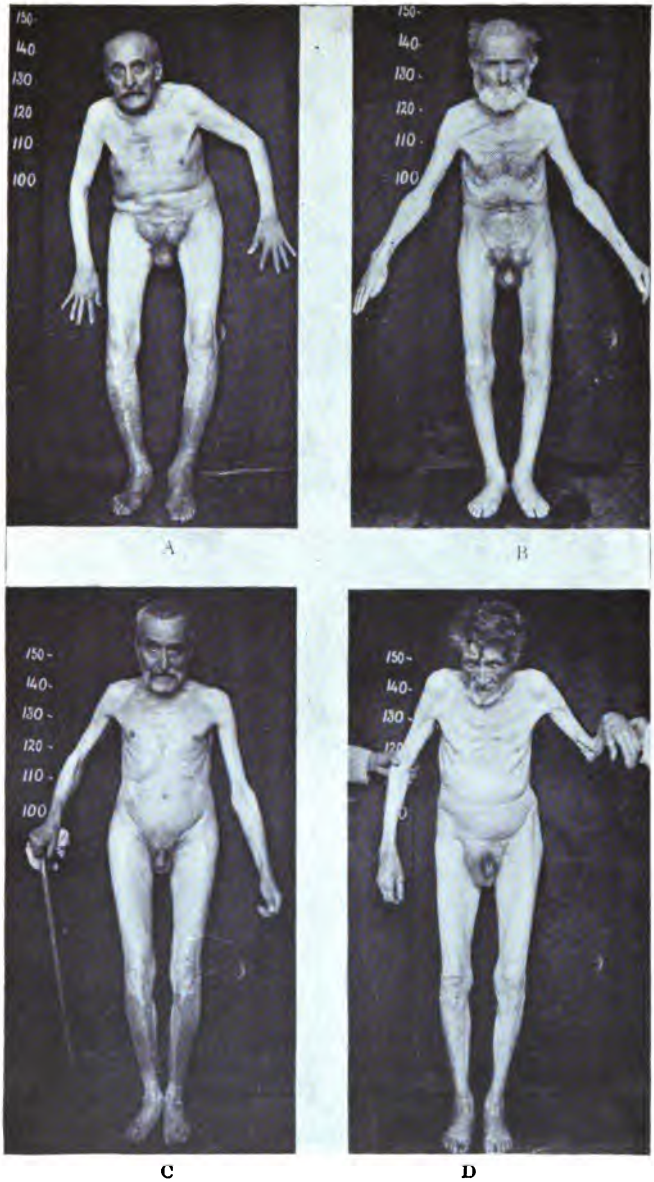


Plate XI.—Senile deformities of the skeleton simulating Paget's Disease.—Nouvelle Iconographie de la Salpêtrière, Jan.-Feb., 1905.

When the feet are brought together the tibiæ are 18 cm. (7 inches) apart. The femora and tibiæ are increased in bulk and curved outward. The tibial crest is smoothed away. The hands and feet are normal.

In examining into the occupations of the cases reported Oettinger and Agasse-Lafont find that about half of those whose occupations were given had to do with acids. They, therefore, suggest that if rachitis be regarded as due to an autointoxication from some organic acid, presumably lactic, that Paget's disease may also be due to an acquired intoxication from an inorganic acid. Experiment has at least proved that the alkalinity of the blood is diminished by the ingestion of hydrochloric acid.

PSEUDO-PAGET DISEASE.

Mocquot and Moutier[1] report upon a set of senile deformities of the skeleton simulating Paget's disease. Pierre Marie first noticed these cases, being attracted by the absence of any increase in the volume of the bones of the lower extremity; indeed, there was rather a diminution of volume and a greatly increased fragility. Further study of these cases showed other important differences. Most of the patients are between 70 and 80 years of age. They showed a marked arching forward of the trunk, well illustrated in Plates X and XI. The head is held well forward; the abdomen shows transverse folds at the umbilicus; the knees are slightly flexed and the legs spread apart. The hands are held out from the body. The deformities are confined practically to the trunk and lower extremities, the head escaping. The deformities of the thorax are especially accentuated, the angle of Louis in the sternum being very prominent. Below, the thorax seems to be forced down into the abdomen. The insertions of the second ribs at the angle of Louis are very evident so that as a rule a groove crosses the thorax at this point, well shown in the figures. In the abdomen there is seen a rounded eminence in both the epigastrium and hypogastrium, with deep transverse folds above the umbilicus

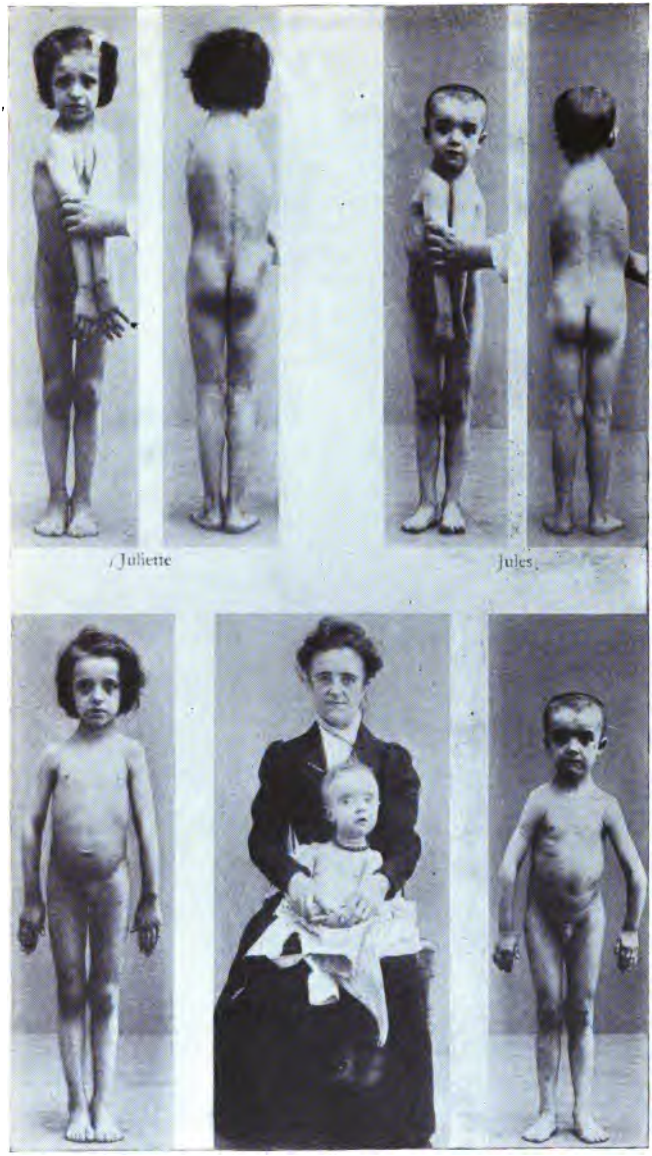
(1) Nouvelle Icon. de la Salpêtrière, January and February, 1905.

ending laterally in depressions in the hypochondriac areas, and a diminution or even disappearance of the space between the ribs and the crests of the ilia. The spinal column shows only one curve, the convexity being posterior. The normal lumbar lordosis is almost entirely wiped out. The accompanying lateral and posterior views illustrate the conformation of the back and the legs as seen from behind. When the heels are held together, as in Fig. F, Plate XI, the internal femoral condyles are seen to be far apart. The underlying factor is evidently osteoporosis, and ordinary bony atrophy. Indeed, the name "pseudo-Paget disease" is not justified, for it is merely a syndrome and not a disease at all.

HEREDITARY CRANIO-CLAVICULAR DIOSTOSIS.

Maurice Villaret and Louis Francois[1] describe a family of four showing a very curious deformity described for the first time by Pierre Marie and Sainton in May, 1897, and May, 1898, under the name above mentioned. The chief characteristics are an exaggerated development of the transverse diameter of the head, delay in the ossification of the fontanelles, aplasia more or less pronounced of the clavicles, and hereditary transmission of the defects. The present series consists of a mother and her three children, her parents being full cousins. The accompanying Plate XII excellently illustrate the deformities of head and arms. The mother had a left clavicle, but the right consisted of only two fragments, an internal and external one. The scapulæ were winged in aspect. The eldest child, Juliette of the figures, shows a bilateral and symmetric deformity of the clavicle, there being merely an internal fragment. The coracoid process of the scapupla is normal. The shoulders droop somewhat. The head is large, the forehead rounded, the facial parts small. The palate is arched and shows a punctiform cavity but no perforation in the midline. Dentition is tardy and complete ossification of the head was delayed. Similar deformities are shown in Jules and Lucienne. The case of Couvelaire is also illustrated in Plate

(1) Nouvelle Icon. de la Salpêtrière, May-June, 1905.



Juliette. The Mother and Lucienne. Jules.

Plate XII.—Hereditary Cranio-clavicular Dysostosis in a mother and three children.—Nouvelle Iconographie de la Salpêtrière, May-June, 1905.

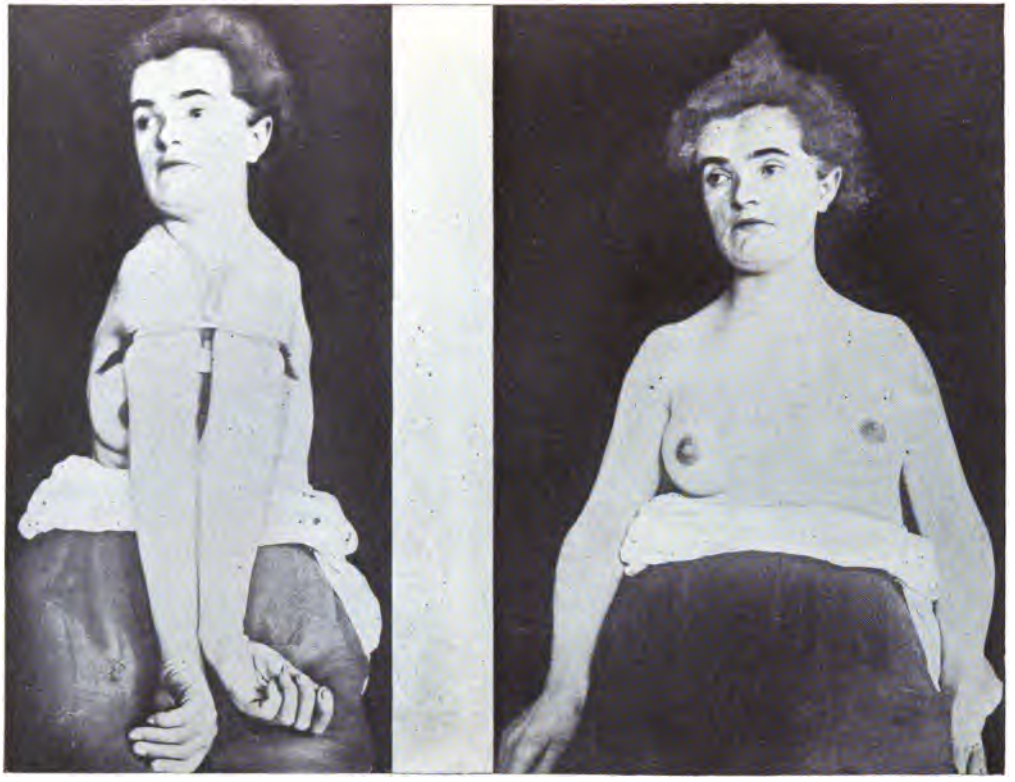


Plate XIII.—Hereditary cranio-clavicular Dysostosis. Couvelaire's Case.—Nouvelle Iconographie de la Salpêtrière, May-June, 1905.

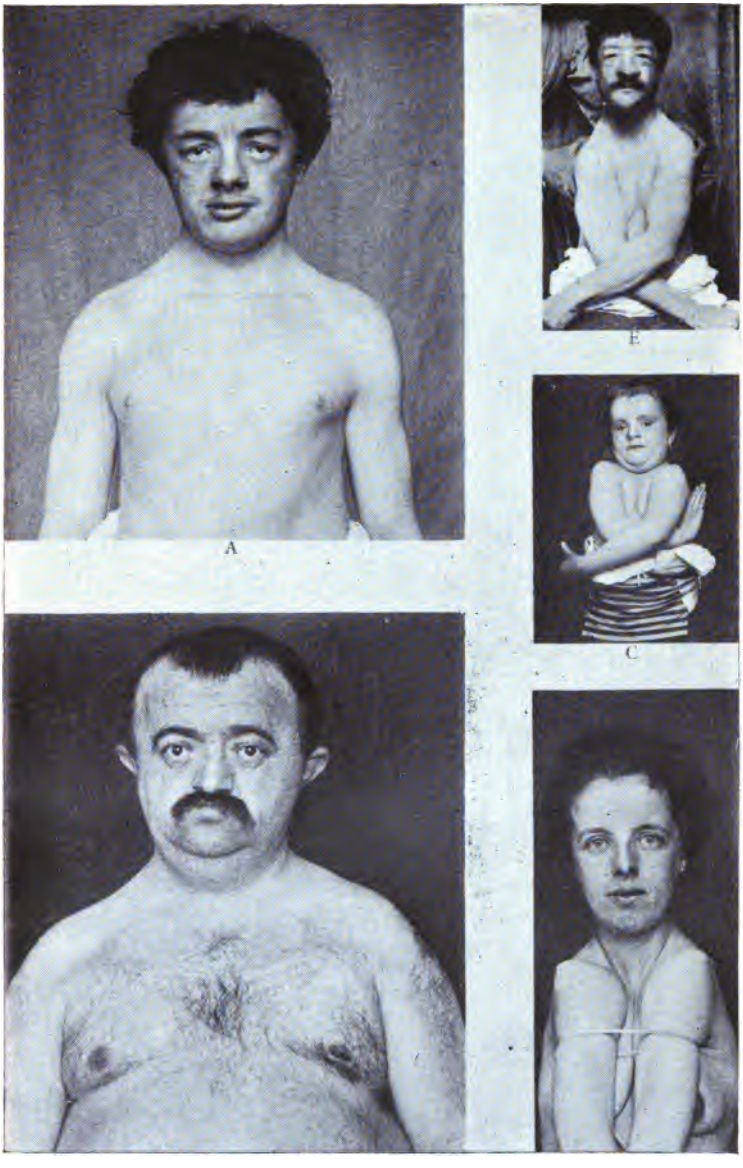


PLATE XIV.—Hereditary cranio-clavicular Dysostosis, A, B, C, D.—Cases of Pierre Marie and Sainton. E.— Hirtz's Case. —Nouvelle Iconograph' de la Salpêtrière, May-June, 1905.

XIII. In all of these cases no defect of intelligence or of sensation or motion has been detected. Couvelaire's case was 4 feet 8 inches tall, the same height as the mother of Juliette, Jules and Lucienne. Other interesting cases are figured in Plate XIV from the reports of Marie and Sainton, and of Hirtz. In all twenty-eight cases are collected from the literature in addition to the four here reported by Villaret and Francois.

MENTAL DISEASES.

SYPHILIS AND GENERAL PARALYSIS.

Christian[1] is not willing to agree that general paralysis is of a syphilitic origin, although he recognizes that he is in a hopeless minority, receiving only the disdainful pity accorded to one possessed of a fixed idea. Yet the only argument bolstering up the syphilitic theory is the statistical one. No set of statistics has, however, been collected in which a few cases are recorded where it has been impossible to demonstrate the existence of syphilis. And yet these exceptional cases cannot be distinguished either clinically or pathologically from the so-called syphilitic ones. Evidently syphilis cannot be the only cause, nor the essential cause. If general paralysis can develop independently of syphilis, it is ridiculous to call it a parasyphilitic disease, as does Fournier. Moreover, statistics furnish merely a *post hoc propter hoc* sort of argument. Hence, the mercurial treatment, universally regarded as unsatisfactory, is worse than useless, and Fournier's proposal to treat every case of syphilis for ten years by periodic onslaughts with mercury is absurd unless it is proved that all syphilitics are apt to become cases of general paralysis. At present, however, there are vastly more syphilitics than paretics in the world.

David Orr[2] investigated the spinal cords of six cases of general paralysis with various complications, such as bedsores, right-sided pyopneumothorax, staphylococcus infection and convulsions, and two cases of acute general paralysis. The spinal cords were treated by the Marchi method, attention being devoted wholly to the posterior columns. His conclusions follow and were constant in all eight cases: "(1) The portion of root between cord and posterior root ganglia shows no degeneration. (2) Degeneration of the internal division of the sensory root is found in its intra-

(1) Progrès Médical, April 29, 1905.
(2) British Med. Jour., Oct. 15, 1904.

medullary path, commencing at the point of entrance into the cord. (3) The external division, or Lissauer's area, remains almost intact. (4) The collaterals are terminals passing into the gray matter share in the degenerative process. (5) The long fibers derived from segments situated low down in the cord are also affected, as shown by the presence and position of the degenerate fibers in Goll's column in the upper parts of the cord." Orr believes that the sensory fibers are specially vulnerable just where they enter the cord, owing to the absence of the neurilemma. At this point, too, the medullated sheath is thinned, and, according to some authorities, is absent. Therefore, toxins, either circulating in the cerebrospinal lymph, or ascending by the nerve sheaths from the limbs, must attack the fibers at this point. As to the question of the nature of the degeneration, the absence of any change in the roots between the ganglia and the cord entirely excludes the view that the above-described appearances are secondary to nerve-cell destruction or to any lesion of the nerves in their extra-medullary path. The degeneration is, therefore, a primary one. These areas affected coincide anatomically both as regards position in the columns and as regards the degeneration of the terminals and collaterals in the gray matter, with those observed in tabes dorsalis. Orr believes that in tabes the chronic affection of the sensory fibers observed is due to the primary influence of a toxin in the posterior lymph system.

O. Fischer[1] discusses cytodiagnosis in progressive paralysis, and decides adversely concerning its value. He calls attention to the fact that positive cases repeatedly examined often fail to show any lymphocytosis of the cerebrospinal fluid, and he illustrates by citing one of his own cases where even shortly before death the cerebrospinal fluid was found to be normal, yet the post mortem findings were pronounced. In this case, however, the meninges showed a great deal of thickening from connective tissue formation, but there was only a slight cellular infiltration. Another case is cited showing a cerebrospinal lymphocytosis and a markedly cellular meningeal infiltration. Fischer, therefore, concludes that the only thing indicated by a cerebrospinal lymphocytosis is the degree of cellular

(1) Prag. med. Woch., Oct. 6, 1904.

infiltration of the meninges, and absence of lymphocytosis does not indicate healthy meninges. Further study showed that the cerebrospinal lymphocytes were identical with those of the cerebral meningeal infiltration.

Gaupp[1] discusses the *prognosis* of general paresis, which he regards as uniformly fatal. Of 175 cases at the Heidelberg clinic, the average duration of life in the chronic dementing form was two and a half years, while in the expansive form the duration was even less. Then 15 per cent of the chronic dementing form were alive after six years, whereas only 7 per cent of the latter group lived more than five years. He is skeptical about the existence of true general paresis for fifteen or twenty years without a fatal issue. Remissions in the chronic dementing form are less common than in the classical type. If speech disturbances are marked the prognosis is worse. The juvenile and senile forms outlast the ordinary cases. Gaupp thinks that many of the so-called recoveries are cases of manio-depressive insanity, alcoholism, katatonia or hysterical degeneracy, and not paresis. It is true that there are remarkable remissions, including the return of the light reflex, the reappearance of a lost knee-jerk and a return of the speech to normal. Even the mental disturbance may clear up completely. A disappearance of all the physical symptoms always carries with it marked mental improvement. Not more than 10 per cent of cases show remissions, and only about 1 per cent show intermissions. The remissions may last from a few months to several years. They are much more apt to occur in cases in which the onset is acute than in those in which it is gradual. Remissions also occur in the expansive, circular and katatonic forms, less commonly in the chronic dementing type. The cases of so-called paresis which are apparently stationary for years are really instances of diffuse cerebral syphilis, alcoholic or traumatic dementia, and dementia precox. Dementia paralytica is a progressive disease.

Charles L. Dana[2] writing on the curability of early paresis is convinced that paresis in its earliest stages is a disease that sometimes may be arrested. This early stage

(1) Deutsche med. Woch., 1904, Nos. 4 and 5. (Also Boston Medical and Surgical Journal, Nov. 24, 1904.)
(2) Jour. Am. Med. Assn., May 6, 1905.

might be called "preparesis." This arrest may be permanent and may be attended with so little mental defect that one may call the patient practically cured. "Tabes and general paresis are congeners, paresis being to the brain what tabes is to the cord, and both being associated in the same patient in 5 per cent of all cases of either, the tabes gradually associating itself with paresis, and *vice versa*. Now if tabes may be arrested in its pretabetic stage, as all freely admit, there seems to be no reason to suppose that paresis may not also be arrested and cured in its earlier stages, and Dana believes that it can be done. He does not assert that paresis when well established can be cured; in fact, he is quite of the contrary opinion. In his "cured" cases it is not a question of remission, because in remissions the mind is by no means restored to its original tone or vigor, whereas in Dana's cases the condition of both mind and body has been restored to a normal level. There is nothing *a priori* impossible in the arrest of paresis, for we are able to arrest degenerative processes in other parts of the nervous system, as well as in the kidneys, liver and other organs.

Dana forfends the claim that he is really reporting cases of cured pseudoparesis, either alcoholic or otherwise. Alcoholic pseudoparesis is only an organic dementia due to connective tissue proliferation, vascular changes and cellular atrophy. Syphilitic pseudoparesis is simply a dementia brought on by syphilitic exudates, leading to more or less severe secondary changes in the meninges and in the vascular supply. "In true paresis the organic changes are comparatively slight at the beginning, and are probably mostly of a parenchymatous nature. There is no exudate and no early gross organic change. It is true, however, that in the early stages of paresis there may be some slight amount of luetic exudate, and that perhaps the degenerative changes in the cell are started by this process. We know also that there are cases of paresis in which there are both true primary degenerative changes and real syphilitic exudates present at the same time, so that the patient may be said to be suffering both from brain syphilis and from a paresis. It is strictly analogous to the conditions which occur in a spinal cord, where at times there may be a true tabetic degeneration and at the same time a decided

syphilitic exudate, so that we have both locomotor ataxia and spinal syphilis together.

"It is at this point that the weakness of my case, as I freely admit, may lie. It may be and probably will be contended that the cases which I assert are in the stage of "præparesis" are really cases only of slight exudative brain syphilis, and that my patients have simply been cured of a slight degree of a perhaps rather diffuse vascular and meningitic exudate. To this I can only reply that I have many times seen precisely this class of cases passing directly into a condition of true paresis, and that in all of my cases where there were admittedly some symptoms of syphilitic exudate, or some kind of gross organic lesion, there were with it also decided psychical and somatic symptoms, such as occur only, or mainly, in connection with the degenerative process of paresis. An Argyll-Robertson pupil, for example, is the sign of the onset of a degeneration, not of exudation. At the very most, while admitting that my opponents may be academically right, I would claim that they are practically wrong, for the reason that, basing my argument on experience with other cases, I feel sure that all or nearly all of these patients would have gone into a condition of true paresis if they had been let alone. Thus, when a patient who has given a distinct history of syphilis develops a form of agitated melancholia, and at the same time shows signs of cerebral degeneration, like the Argyll-Robertson pupil and disturbances in the knee reflexes, I should certainly be apprehensive that, under ordinary conditions, he would eventually develop a paresis, for I have seen a number of patients who entered paresis through this peculiar gate of melancholia, with somatic signs as indicated.

"Again, if a patient with a history of syphilis, after a certain period begins to develop convulsions and shows Argyll-Robertson pupils, exaggerated reflexes, then begins to develop symptoms of loss of memory, change of character and disturbances of the instinctive feelings, I should feel very certain that if left alone he would pass into the condition of paresis; for it is through the gate of convulsive disturbances, epileptiform seizures and peculiar somatic signs that paresis sometimes develops."

"Still further, when a patient, who gives perhaps a doubtful history of syphilis but whose life is such that he might easily have been subjected to it, and who has a headache, an eye-palsy, and previous to that has for some time shown great extravagance in action and ideas, with a decided change in character and weakness of memory, I would here also feel very sure that a paresis was developing.

"Having observed the total disappearance of all these symptoms, under treatment, and the restoration of the patients practically to their former health, it has seemed to me that I may be right in claiming that it is possible to arrest for an indefinite time a disease which is certain to become a general paresis."

To substantiate his affirmation Dana recounts the histories of 7 such arrested or cured cases. As to treatment, advice is given to turn the patient at once from his mode of living. He should be sent away for rest and fresh air, and he should be given hypodermic injections of the bichlorid or salicylate of mercury, accompanied or followed by iodid of potassium and tonic measures. One-fourth of a grain of either of these salts of mercury given twice a week is sufficient, and this treatment may be kept up two or three months. In other cases two or even three grains once or twice a week are required. The technic requires care. During the course of the treatment there should be a very liberal use of lukewarm and hot bathing (a warm bath every day and a hot bath once or more weekly), and every possible attention should be given to the general nutrition of the patient. "In spite of the general skeptical and critical tone of the discussion of Dr. Dana's paper, it is noticeable that few if any of those who took part ventured to assert the absolute impossibility of recoveries of the disease in its earlier stages. If Dr. Dana's views are correct, the importance is emphasized of an early diagnosis and treatment, or of at least tentative treatment, even in cases in which such symptoms as the Argyll-Robertson pupil or convulsions indicate decided degenerative changes. We are very far from having reached the last word as to the nature and curability of paretic dementia."[1]

N. F. McHardy[2] reports 2 cases of general paralysis suc-

(1) Journal Am. Med. Assn., May 6, 1905, Editorial, p. 1,450.
(2) British Med. Jour., Jan. 28, 1905.

cessfully treated by urotropin. Most of McHardy's account of these cases is devoted to the effects of urotropin, the symptoms of the patient and physical findings being reduced to a description of four and a half lines in the first case and five and a half lines in the second. [It is extremely doubtful whether either case is general paralysis and whether urotropin has the slightest effect upon that disease.—ED.]

DEMENTIA PRECOX.

Quite the best general article which appeared upon dementia precox during the past year was the one by M. Deny,[1] read at the Fourteenth Congress of Alienists and Neurologists at Pau. According to Deny, dementia precox is a psychosis essentially characterized by a *special and progressive weakening of the intellectual faculties* (the italics are the author's own), as a rule developing in young people, normal up to the onset of the disease; and it is accompanied by *various psychic disturbances* such as excitation, depression, confusion, delirious conceptions, hallucinations, etc. It terminates in the great majority of cases in *complete loss of every sort of physical and psychical activity.* The onset is ushered in by all sorts of nervous disturbances, followed by attacks of delirium, which, although polymorphous, have certain characteristic features about them and are accompanied by special physical signs The symptoms of dementia precox are divisible, therefore, into two groups: the psychic and the physical. Among the former are two groups, the constant, invariable and fundamental symptoms on the one hand, and the episodic, variable and accessory ones on the other.

(I) The fundamental psychic symptom is the specific dementia which involves every phase of psychic activity. This dementia is first of all a *primary* one, antedating all other symptoms. Secondly, the dementia is an all-embracing one, affecting the three great psychic faculties (feelings, intelligence and will). Yet it is *elective,* since it does not affect all three of these faculties equally or in the same manner. For example, the intellectual defect in dementia

(1) Revue Neurologique, Aug. 30, 1904.

precox is incomplete, though never absent in the onset. It is usually the first of the three faculties to be involved, the affective and moral sentiments or feelings next yielding, and ultimately the voluntary activity being seriously impaired or lost. When the feelings and will are lost, then the intellectual deficit goes on to totality. Thus dementia precox differs from general paralysis and senile dementia. If one were to exaggerate in an epigrammatic manner the special characteristic of each of these three dementias, one might say that dementia precox at the beginning is especially *moral,* dementia paralytica is especially *intellectual,* and senile dementia is especially *voluntary.*

From the *affective and moral standpoint,* there are to be found slight modifications of character, such as a changeability of mood, instability, a tendency to revery and isolation, and irritability. There follow in turn apathy, moral dulness and emotional indifference betraying themselves in untidiness in clothing and manners, a loss of all love for family and friends, disregard for the conventionalities, a lack of interest and curiosity. This change in the affective sensibilities in those who manifest no interest in their surroundings, yet remember fairly accurately, constitutes almost a pathognomonic sign of dementia precox.

From the standpoint of the *will* there is a diminution in voluntary motor activity (abulia), and the will power is incapable of exercising any inhibitory influence on automatic movements. When the *katatonia syndrome* is present, there are: (1) signs of negativism, slowness and hesitation of movement on account of psychic restraint, an invincible resistance to motor solicitations, complete inertia and stupor. (2) There are also signs of *suggestibility* in the extreme docility, initiative activity, waxy flexibility of the limbs, cataleptiform attitudes, automatic continuation of movements, echolalia, and echopraxia. (3) There are lastly, signs of *stereotypy* characterized by the incessant repetition of the same motions, acts or words. A continuous necessity for movement, disorderly gesticulations, sudden impulses, explosions of laughter or of crying, all indicate, on the other hand, the exaggeration of automatic activity.

From the *intellectual* standpoint, the disturbances chiefly involve *attention* (prolongation of the time of reac-

tion), the *memory* (impossibility of fixing new images correctly, verbigeration in both speaking and writing, formation of new words), and the *association of ideas.*

(II) The accessory psychic symptoms and signs. If the specific dementia exists all by itself during the whole course of the affection, without any true delirious conceptions, hallucinations and marked phenomena of excitation, the case is to be looked upon as a mild one or as a *forme fruste.* The other clinical varieties of the disease, katatonia, hebephrenia and the paranoiac form of dementia precox, result from the association of dementia precox simple with other less important psychic symptoms, such as: (a) Delirious manifestations. Most often there are delusions of grandeur, but there may be hypochondriac, mystic or erotic ideas and delusions, without hallucinations; yet all these concepts are polymorphous, unsystematized, equalling if not surpassing the singular concepts of paralytic dementia. Still, in some forms, as the paranoiac, the delirium is coherent, consistent and insistent, being focussed upon some idea of persecution or grandeur.

(b) Sensory manifestations. These are chiefly hallucinations of hearing, vision, or general sensibility.

(c) States of excitation, depression and katatonic stupor. The delirium and the sensory disturbances predominate in the hebephrenic and paranoid forms. In the katatonic form there are to be found the most varied psycho-motor reactions. Sometimes there is simultaneously present *intellectual excitability* (irrepressible loquacity, incoherent verbiage, jargon, verbigeration, meaningless declamation of words and phrases grammatically correct but completely devoid of sense), and *motor excitability* (attitude of the sphinx, of an athlete, of the crucifixion, draping of the body in the classical manner, sudden gesticulations, dancing, etc.). Sometimes there is, on the other hand, *stupor* in the katatonic form, the patient remaining motionless all day long, stiff, often in a painful attitude; negativism, but sometimes for a few instants suggestibility. The only explanation for the katatonic symptoms of dementia precox is to be found in the progressive enfeeblement of all the intellectual operations.

(III) Physical signs. These are much less characteristic than those found in dementia paralytica. Those most

frequently observed are: exaggeration of the tendon-reflexes; diminution of the plantar cutaneous reflexes; pupillary disturbances such as diminution of the reaction to light or distance, inequality of the pupils, mydriasis and myosis; diminution of the pharyngeal and conjunctival reflexes; disturbances of menstruation; vaso-motor disturbances, such as cyanosis, coldness of the extremities, and hyperidrosis; and dermographia. There seems to be a urinary formula, too, in dementia precox, consisting of oliguria, hypoazoturia, hypophosphaturia, hypochloruria; but this formula doubtless varies during the different periods of the disease. The elimination of methylen blue is retarded and prolonged.

Dementia precox must be considered as a clinical entity, as yet without an indisputable pathologic basis. The meninges are not altered (there are no cells constantly to be found in the cerebrospinal fluid), there are no morphologic changes in the gyri and sulci of the brain. In the brain and cord, lesions limited to the neurones of the association-centers alone have been described, such as atrophy of the large pyramidal cells except in the motor cortex, with pigmentary degeneration and diffuse chromatolysis of the same cells.

As to the frequency of dementia precox, it may be said to comprise about one-fourth of the asylum patients. The period of life most favorable for its development is from 15 to 30 years of age, but certain cases of katatonia and paranoid dementia may appear only in adults and even as late as the menopause. The two sexes are about equally affected. Hereditary neuro-psychopathy is found in about 70 per cent of the patients affected. On the other hand, physical stigmata of degeneration are rarely observed. Want, deprivation, physical and moral exhaustion, puberty, menstrual disturbances, the puerperal state, all have an incontestible influence upon its development, and they seem to justify the hypothesis of an autointoxication, perhaps of sexual origin. Beneath all these causes there is doubtless an underlying special if not specific cause of this mental malady.

"Is dementia precox a *constitutional* psychosis, inherent to the individual, of which heredity is the necessary and sufficient cause, or is it, on the contrary, an *accidental*

psychosis whose development is bound up with powerful etiologic factors other than predisposition, such as material lesions suddenly or gradually developed? From its clinical history its etiology, the fact that it assails a person mentally normal up to the time of its appearance, one would be apt to regard it as an accidental malady. Indeed, one may conclude that dementia precox, while subject to the same limitations as general paralysis to the inevitable law of inherited and acquired predisposition, is an accidental malady in the same manner and to the same degree as dementia paralytica."

A very extensive discussion (*) followed, in which it appeared that many of the French psychiatrists, notable Regis, were unable to subscribe to Deny's conception of the disease, fault being found in his conclusion that the disease is accidental rather than constitutional.

Conolly Norman[1] believes that Kraepelin's grouping of hebephrenia, katatonia, and the paranoid forms makes so vast a congeries that it seems impossible to perceive any connecting link between the items of the mass, save in their origin at the age of adolescence and in their supposedly unfavorable termination. To Norman katatonia and hebephrenia both have considerable value as types of mental disease, with well-marked signs and facies. A good case of katatonia is so distinct in its mental and physical symptoms that it seems almost a specific disease. The studies of Bruce and others would justify such an opinion, were it not for the difficulty interposed by intermediate forms. It is this only which might justify the grouping of hebephrenia and katatonia as dementia precox.

The chief difficulty is in the paranoid forms. There is absolutely no criterion by which these can be distinguished from paranoia, unless we hold that nothing can be called paranoia except the most exquisitely pure and typical cases of that disease. It is difficult to believe in a disease so poorly differentiated as yet. At present everything is being dubbed "dementia precox." Indeed, both of the terms, "dementia" and "precox," are faulty, for first there is no dementia, but merely an involvement of the intellectual,

*Comprising twelve pages of fine print in the Revue Neurologique, Aug. 30, 1904.

(1) British Medical Journal, Oct. 15, 1904.

affective and voluntary faculties, and, secondly, cases developing at the menopause or later are hardly precocious.

F. X. Dercum[1] reviews the early history of the dementia precox concept, foreshadowed in the writings of Spurzheim, Esquirol, and Morel. He then develops Kraepelin's conception of dementia precox, finding fault with the word dementia since it implies an absolutely unfavorable prognosis. A much better term would be "insanity of adolescence." Dercum's views are best summarized by himself: "We have in the solar spectrum seven colors, each color passing by a most gradual transition into another. Shall we, because it is impossible to draw hard and fast lines—because we cannot say, for instance, where violet ends and blue begins, say that violet and blue are the same; or that green and yellow—most opposite in their reaction on the human sensorium—are the same because they pass by insensible transition one into the other? Surely nothing can be gained by wiping out well established lines of demarcation. Change does not always mean progress; it sometimes means error. Practical experience teaches us that no sharp differentiation can be drawn between the various forms of dementia precox—hebephrenia, catatonia and dementia paranoides. Every now and then we meet with cases of hebephrenia in which passing motor phenomena, convulsions, transitory rigidity or stereotypy is noted, or again cases of dementia paranoides in which like features cause an approximation to catatonia. Similarly I believe that it is often difficult, if not impossible, to draw a differentiation between a dementia paranoides and a phantastic paranoia, just as it is impossible always to make an absolute differentiation between the latter and the paranoia of Magnam, the *'combinirte'* form of Kraepelin."

(1) Journal Am. Med. Assn., Feb. 4, 1905, p. 355. (The same article in German is to be found in the Wiener klin. Wochenschrift Aug. 7, 1904.)

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